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Office of Administrative Law Judges
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Issue Date: 20 January 2005

Case No. 2002-BLA-140

In the Matter of:
ERNEST E. BALSLEY,
Claimant,

v.

PEABODY COAL COMPANY,
Employer,
and
OLD REPUBLIC INSURANCE CO.
Carrier,

and
DIRECTOR, OFFICE OF WORKERS'
COMPENSATION PROGRAMS,
Party-in-Interest.

APPEARANCES:
Christopher R. McFadden, Esq.
On behalf of Claimant

Scott A. White, Esq.
On behalf of Employer

BEFORE: THOMAS F. PHALEN, JR.
Administrative Law Judge

DECISION AND ORDER – AWARD OF BENEFITS

This is a decision and order arising out of a claim for benefits under Title IV of the Federal Coal Mine Health and Safety Act of 1969, as amended by the Black Lung Benefits Act of 1977, 30 U.S.C. §§ 901-962, ("the Act") and the regulations thereunder, located in Title 20 of the Code of Federal Regulations. Regulation section numbers mentioned in this Decision and Order refer to sections of that Title.¹

¹ The Department of Labor amended the regulations implementing the Federal Coal Mine Health and Safety Act of 1969, as amended. These regulations became effective on January 19, 2001, and are found at 65 Fed. Reg. 80, 045-80,107 (2000)(to be codified at 20 C.F.R. Parts 718, 722, 725 and 726). On August 9, 2001, the United States District Court for the District of Columbia issued a Memorandum and Order upholding the validity of the new regulations. All citations to the regulations, unless otherwise noted, refer to the amended regulations.

On October 26, 2001, this case was referred to the Office of Administrative Law Judges by the Director, Office of Workers' Compensation Programs, for a hearing. (DX 33).² A formal hearing on this matter was conducted on January 28, 2004, in Cincinnati, Ohio by the undersigned Administrative Law Judge. All parties were afforded the opportunity to call and to examine and cross examine witnesses, and to present evidence, as provided in the Act and the above referenced regulations.

ISSUES³

The issues in this case are:

- 1) Whether the miner has pneumoconiosis as defined by the Act;
- 2) Whether the miner's pneumoconiosis arose out of coal mine employment;
- 3) Whether the miner is totally disabled;
- 4) Whether the miner's disability is due to pneumoconiosis;
- 5) Whether the miner has one dependent for the purpose of augmentation; and
- 6) Whether the evidence establishes a material change in conditions under §725.309(c), (d).

(DX 33; Tr. 13-14).

Based upon a thorough analysis of the entire record in this case, with due consideration accorded to the arguments of the parties, applicable statutory provisions, regulations, and relevant case law, I hereby make the following:

FINDINGS OF FACT AND CONCLUSIONS OF LAW

Background

Ernest Balsley ("Claimant") was born on June 7, 1937 and was 66 years old at the time of the hearing. (DX 1; Tr. 16). Claimant testified that he has a ninth grade education. (DX 1). Claimant married Shirley (Searles) Balsley on June 15, 1991. (DX 1, 6; Tr. 16). Also, Claimant was formerly married to Norma Jean Vansickle from November 11, 1983 until their divorce on February 23, 1989. (DX 1). He reported that he does not make court ordered support payments to Ms. Vansickle, nor does he make substantial contributions to her. (DX 1). Finally, Claimant

²In this Decision, "DX" refers to the Director's Exhibits, "EX" refers to the Employer's Exhibits, "CX" refers to the Claimant's Exhibits, and "Tr." refers to the official transcript of this proceeding.

³At the hearing, Employer withdrew as contested the issues of whether the claim was timely filed; whether the miner worked after December 31, 1969; and whether the named employer is the Responsible Operator. (Tr. 13). Additionally, at the hearing the parties stipulated to a length of coal mine employment of at least 12 years. (Tr. 13). Finally, Employer listed other issues that will not be decided by the undersigned; however, they are preserved for appeal. (Tr. 13; Item 14 DX 33).

has one dependent child, Philip (Todd) Balsley, who is disabled and over the age of 18. (DX 1; Tr. 38). Claimant stated on his application for benefits that Todd, who lives with another of Claimant's former wives, Easter L. Haines, receives Social Security from Claimant's account. (DX 1). But, at the hearing, Claimant testified that Todd lives with him and his wife, and that Claimant provides financial support beyond what his son receives from Social Security. (Tr. 38). Since the hearing took place more than three years after Claimant's application, in which time a change in the residence of Claimant's son has apparently transpired, and there is no evidence in the record to contest Claimant's hearing testimony, I find Claimant's testimony to be credible. Therefore, I find that Claimant has two dependents for purposes of augmentation.

On his application for benefits, Claimant stated that he engaged in coal mine employment for 12 years. (DX 1, 2). Claimant's coal mine employment was working as a general inside laborer and trackman. (Tr. 17-18). As a general laborer, his job included rock dusting setting timbers, pinning top, building cribs, shoveling the belt line, drilling, shooting, driving a buggy, acting as beltman, acting as a pinner man, and serving as a miner helper. (DX 4, 5; Tr. 25-30). As a trackman he was responsible for laying track to transport miners too and from the mine. (DX 4; Tr. 18). Claimant explained that the physical requirements varied depending on the task, with some jobs requiring him to lift 35-50 pounds. (DX 5). Also, the trackman position required him to assist another man in loading 25-30, 200-pound ties per day, and to drive in 20 to 26 spikes with a sledgehammer. (Tr. 21, 39). While he stated that his last position was as a trackman, he also claimed that these positions tended to overlap. (DX 4). Claimant last worked in and around coal mines in 1987 when he was laid off. (DX 1, 4). Next, Claimant worked as a truck driver for Greenlawn Mobile Homes until quitting in 1990 after suffering a stroke. (DX 2, 5; Tr. 33, 41). He has not filed, nor does he receive any state or Federal Workers' Compensation benefits based on his disability due to coal workers' pneumoconiosis ("CWP"). (DX 1).

Procedural History

Claimant filed his first claim for benefits on January 23, 1991. (DX 31). It was denied by the District Director, Office of Workers' Compensation on July 12, 1991. The record reflects no further action on this claim.

Claimant refilled on August 9, 1994. (DX 32). The Director initially denied this claim on November 9, 1994, finding that Claimant failed to establish the presence of pneumoconiosis, or that the disease was caused by coal mine employment. A conference was held, and on May 26, 1995, the Director issued a Memorandum of Informal Conference, finding that Claimant had only satisfied the total disability element. The director affirmed this decision on August 8, 1995. The record reflects no further action on this claim.

Claimant filed his instant claim for benefits of November 1, 2000. (DX 1). On May 14, 2001, the Director initially denied this claim. (DX 18). Claimant appealed the decision on June 18, 2001. (DX 20). Due to Director's inability to hold an informal conference, he re-categorized the case as a hearing, and forwarded it to this office. (DX 26, 33). The hearing was held before the undersigned on January 28, 2004.

Length of Coal Mine Employment

Claimant was a coal miner within the meaning of § 402 (d) of the Act and § 725.202 of the regulations. On a Black Lung Claim Employment Inquiry from, Peabody Coal Company's Employment Relations Representative, Mike Turner, reported that Claimant was employed by Peabody from January 28, 1975, until October 9, 1987. (DX 4). Also, on both his application for benefits and his employment history form, Claimant stated that he engaged in coal mine employment for 12 years. (DX 1-2). Furthermore, the parties stipulate to at least 12 years of coal mine employment. (Tr. 13). A review of the record supports this stipulation. Therefore, I find that Claimant engaged in qualifying coal mine employment for at least 12 years.

Claimant's last employment was in the State of Ohio; (DX 4; Tr. 17), therefore, the law of the Sixth Circuit is controlling.⁴

Responsible Operator

Liability under the Act is assessed against the most recent operator which meets the requirements of §§ 725.494 and 725.495. The District Director identified Peabody Coal Company as the putative responsible operator. (DX 15;16). The Employer's attorney did not object when this issue was withdrawn at the hearing. (Tr. 13). Therefore, I find that Peabody Coal Company is properly designated as the responsible operator in this case.

NEWLY SUBMITTED MEDICAL EVIDENCE

X-RAY REPORTS

Exhibit	Date of X-ray	Date of Reading	Physician/Qualifications	Interpretation
EX 10-11	03/26/91	3/13/02	Wiot, BCR ⁵ , B-reader ⁶	Negative
EX 15-16	03/26/91	5/24/02	Spitz, BCR, B-reader	Negative
EX 10-11	10/13/94	3/13/02	Wiot, BCR, B-reader	Negative
EX 15-16	10/13/94	5/24/02	Spitz, BCR, B-reader	Negative

⁴ Appellate jurisdiction with a federal circuit court of appeals lies in the circuit where the miner last engaged in coal mine employment, regardless of the location of the responsible operator. *Shupe v. Director, OWCP*, 12 B.L.R. 1-200 (1989)(en banc).

⁵ A physician who has been certified in radiology or diagnostic roentgenology by the American Board of Radiology, Inc., or the American Osteopathic Association. *See* 20 C.F.R. § 727.206(b)(2)(III). The qualifications of physicians are a matter of public record at the National Institute of Occupational Safety and Health reviewing facility at Morgantown, West Virginia.

⁶ A "B" reader is a physician who has demonstrated proficiency in assessing and classifying x-ray evidence of pneumoconiosis by successful completion of an examination conducted by or on behalf of the Department of Health and Human Services. This is a matter of public record at HHS National Institute for Occupational Safety and Health reviewing facility at Morgantown, West Virginia. (42 C.F.R. § 37.51) Consequently, greater weight is given to a diagnosis by a "B" Reader. *See Blackburn v. Director, OWCP*, 2 B.L.R. 1-153 (1979).

DX 14	01/09/01	01/10/01	Hackett	Bronchitis with COPD ⁷
DX 13	01/09/01	03/23/01	Cohen, B-reader	Negative
DX 12	01/09/01	04/19/01	Sargent, BCR, B-reader	Negative
DX 28	01/09/01	10/02/01	Wiot, BCR, B-reader	Negative
EX 1-2	01/09/01	11/20/01	Spitz, BCR, B-reader	Negative
EX 3	01/09/01	12/13/01	Shipley, BCR, B-reader	Negative
EX 5, 6	01/09/01	01/02/02	Renn, B-reader	Negative
EX 7	01/09/01	02/28/02	Fino, B-reader	Negative
DX 23	08/15/01	08/15/01	Altmeyer, B-reader	0/1 tt
DX 29	08/15/01	08/15/01	Benson	Abnormal ⁸
EX 29	08/15/01	02/19/04	Wiot, BCR, B-reader	Negative
EX 6, 28	08/15/01	02/26/04	Renn, B-reader	Negative

PULMONARY FUNCTION STUDIES (“PFT”)

Exhibit/ Date	Co-op./ Undst./ Tracings	Age/ Height⁹	FEV₁	FVC	MVV	FEV₁/ FVC	Qualifying Results
DX 8 1/9/01	Good/ Good/ Yes	63 71”	1.09 1.26*	2.85 3.21*	29	38 39*	Yes Yes* ¹⁰

⁷ This diagnosis by the Fantus Clinic’s department of radiology doctor did not utilize the DOL form CM-33, and as a result, the report did not make a determination on the presence of opacities necessary to determine clinical pneumoconiosis, and it did not make a determination concerning film quality. As a result, the report is not usable under §718.212(a)(1) for making a determination of clinical pneumoconiosis.

⁸ Dr. Bensen’s report did not utilize the DOL form CM-33, and as a result, the report did not make a determination on the presence of opacities necessary to determine clinical pneumoconiosis, and it did not make a determination concerning film quality. As a result, the report is not usable under §718.212(a)(1) for making a determination of clinical pneumoconiosis.

⁹ I must resolve the height discrepancy recorded on the pulmonary function tests. *Protopappas v. Director, OWCP*, 6 B.L.R. 1-221 (1983). Since DX 8 and 24 are reports by examining physicians for the purpose of identifying total disability due to pneumoconiosis, and DX 11 is merely a treatment record, I accord DX 8 and 24 more weight. Therefore, I find that the miner’s actual height is 71 inches.

¹⁰ Dr. Richard A. Katzman determined the vents in the January 9, 2001 PFT to be acceptable. (DX 9).

DX 24 8/15/01	Good/ Good/ Yes	64 71"	.82 1.08*	2.21 2.97*	32 45*	37 36*	Yes Yes*
EX 24 11/19/02	Good/ Not listed/ Yes	65 65.5"	1.20 1.27*	2.75 *2.94	37	44 43*	Yes Yes*

*post-bronchodilator values

ARTERIAL BLOOD GAS STUDIES

Exhibit	Date	pCO₂	pO₂	Qualifying
DX 8, 11	1/09/01	34.8	68.7	No
DX 24	8/15/01	37.1	62.5	No

All values are pre-exercise

ABG STUDIES FROM TREATMENT RECORDS

Exhibit	Date	pCO₂	pO₂	Qualifying
EX 17	10/15/90	44.2	66.6	No
EX 17	11/01/90	49.78	93.8	No
EX 17	11/01/90	44.2	67.8	No
EX 17	11/01/90	42.8	44.8	Yes
EX 17	01/25/91	36.3	127.5	No
EX 17	01/25/91	28.1	168.1	No
EX 17	01/25/91	37.1	49.1	Yes
EX 17	01/25/91	28.7	92	No
EX 17	01/26/91	35.3	103.7	No
EX 17	01/26/91	38.1	84.2	No
EX 17	01/26/91	38.7	80.4	No
EX 17	01/26/91	39.1	79.9	No
EX 17	01/26/91	39.4	73.4	No
EX 17	01/27/91	41.4	79.9	No
EX 17	01/27/91	41.9	89.4	No
EX 17	01/27/91	42.7	134.1	No
EX 17	01/28/91	40.4	88.6	No
EX 17	01/28/91	38.2	53.4	Yes
EX 17	01/31/91	42.7	54.6	Yes
EX 17	02/04/91	35.5	61.7	Yes
EX 13	07/16/91	41.2	77.9	No
EX 17	03/01/92	Illegible	Illegible	N/A
EX 17	04/09/92	38.6	79.2	No
EX 17	10/06/93	38.3	74.4	No

EX 17	11/04/93	41	71.9	No
EX 17	08/02/94	42	71.5	No
EX 17	01/06/95	44	100.8	No
EX 17	01/09/95	42	66.5	No
EX 17	02/13/95	42.5	58.9	Yes
EX 17	04/11/95	37.6	61.2	Yes
EX 17	09/02/95	33.3	65.3	Yes
EX 17	09/28/95	30.1	60.7	Yes
EX 17	10/03/95	43.9	65.2	No
EX 17	11/06/95	31.6	61.7	Yes
EX 17	02/22/96	38.7	63.5	No
EX 14	09/11/96	39	72	No
EX 14	02/23/98	36.8	60.1	Yes
EX 14	10/29/98	39.2	79.2	No
EX 14	12/27/98	36	71.8	No
DX 27	02/26/01	32.3	62.7	Yes
EX 24	02/26/01	64.8	70.4	Yes
EX 24	02/26/01	54.8	75.7	Yes
EX 24	02/26/01	35.6	84.6	No
EX 24	03/02/02	38.5	72.8	No
EX 24	10/27/02	40.7	95.2	No
EX 25	07/17/03	39.6	95	No

All values are pre-exercise

Narrative Medical Evidence

Claimant testified that he was under the treatment of Dr. Paul Mumma at the time of the hearing. (Tr. 43). In a letter dated January 7, 2004, Dr. Mumma, who is certified by the National Board of Osteopathic Medical Examiners and has completed the Federal Licensing Examination, stated that he had been treating Claimant for approximately 18 months for severe COPD. (CX 6). Based on symptomatology (severe incapacitated exertional dyspnea, occasional dyspnea at rest, frequent exacerbation of chronic obstructive pulmonary disease (“COPD”) which has required hospitalization, chronically on oxygen to help ease his shortness of breath, becomes significantly hypoxemic without supplemental oxygen); employment history (12 years in underground coal mines ending in 1987); smoking history (quit smoking in 1980 and has had progressive lung disease since that date, which is excessive for his smoking history); clinical findings (fibrotic disease in excess of what one would expect from tobacco induced obstructive lung disease raising the possibility of particle induced pulmonary fibrosis); numerous bronchoscopies, CT scans, and chest x-rays (severe obstructive lung disease with fibrotic changes ascending what one would expect from COPD); PFTs (severe impairment and diffusion capacity greatly reduced from someone with simple chronic hypoxemia); and ABGs (chronic hypoxemia, when the patient becomes significantly sick he does retain carbon dioxide and becomes acidotic); Dr. Mumma diagnosed CWP with secondary pulmonary fibrosis. He stated that while there is a casual link between tobacco use and obstructive lung disease, he believes the severity and progression of Claimant’s obstructive lung disease is more typical of workers pneumoconiosis

with fibrosis. He opined that a lung biopsy would confirm his findings, but due to Claimant's physical condition, such a procedure should not be performed for legal reasons alone since they would have no clinical utility other than to prove Claimant's case.

Dr. Robert A Cohen, an internist, pulmonologist, and B-reader, examined the Claimant on January 9, 2001, and submitted his report on March 23, 2001. (DX 8, 10-11, 13; CX 2). Based on symptomatology (wheezing, dyspnea, cough, phlegm production, and left hemiplegia), employment history (5 years as a trackman, loading 150 pound ties and using a 16 pound sledgehammer to drive spikes, 12 years total coal mine employment), individual history (wheezing, chronic bronchitis, allergies, diabetes mellitus, high blood pressure, right sided middle cerebral artery stroke in 1990 with residual left hemiplegia, and hospitalized in 1998 for COPD/pneumonia), family history (high blood pressure and heart disease), smoking history (smoked cigarettes from 1953 to 1979 at a rate of 1 pack per day for 23 years and ½ pack per day for 3 years), physical examination (2 cm. diaphragmatic excursion, distant breath sounds, but no crackles and no wheezes), chest x-ray (emphysema but negative for pneumoconiosis), PFT (severe obstructive defect), and an ABG (mild hypoxemia at rest), Dr. Cohen diagnosed COPD caused by cigarette smoking and coal dust exposure, because there are no other significant occupational exposures which could have contributed to Claimant's condition. He speculated that the COPD was "most likely emphysema." Also, he diagnosed hemiplegia as a result of the 1990 CVA. Finally, Dr. Cohen diagnosed severe obstructive lung disease based on an FEV 1 of only 29% of predicted, FEV1 values that improved only slightly with bronchodilators, and a moderately impaired diffusion capacity. After establishing a detailed account of the exertional requirements of Claimant's last position, he concluded that Claimant's degree of impairment would clearly disable him from his last coal mining job which required heavy exertion.

Dr. Cohen also provided a consulting opinion on January 2, 2004. (CX 2). His report included an analysis of the following records: Claimant's coal mine employment; smoking history; past medical history; physical examinations conducted by Dr. Altmeyer on August 15, 2001, Dr. Cohen on January 6, 2001, and Dr. Knight on October 13, 1993 and March 16, 1991; approximately 95 chest x-ray interpretations dated between 1985 and 2003; lung scans dated November 4, 1993 and March 14, 2002; PFT values dated November 19, 2002, May 11, 1995, October 13, 1994, March 26, 1991 and December 30, 1986; ABG studies¹¹ dated August 15, 2001, January 9, 2001, October 13, 1994, and March 26, 2001; consulting opinions from Dr. Tuteur, Dr. Renn, and Dr. Fino; and medical treatment records from Genesis Health Care, Good Samaritan Hospital, Bethesda Hospital, Dr. Mumma, Dr. Albrini, Dr. Kalis, and Dr. Elston, spanning 1983 to 2003. Based on this evidence, Dr. Cohen concluded that Claimant does suffer from CWP, substantially related to 12 years of coal mine employment and 25-30 pack-years of smoking. To support this conclusion, Dr. Cohen first looked to Claimant's history, including the fact that Claimant quit smoking 5-7 years prior to his cessation of coal mining, and that he had symptoms of COPD as noted by several examiners dating back to his first few years in the mines. Next, he looked to objective test results for support. Concerning the physical examination results, he concluded that the results were consistent with chronic lung disease including decreased breath sounds, poor air movement, crackles, rhonchi, wheezing, and rales.

¹¹ There were a large number of ABG studies included in the treating records, but Dr. Cohen stated that these were not as useful for evaluating permanent impairment as the studies done for black lung evaluations because Claimant was suffering from exacerbations at the time the studies were performed.

Also, the PFT results demonstrated severe obstructive lung disease, with one study showing a barely significant response to bronchodilators, and with no study recording an FEV1 value ever rising beyond the level of severe impairment. These studies also showed that Claimant's lung function progressively deteriorated over the years after he stopped mining and smoking. The ABG studies, according to Dr. Cohen, reflect mild hypoxemia at rest which progresses to moderate hypoxemia by August 15, 2001. Finally, Dr. Cohen discounted the negative x-ray results by stating that the film results may be negative in some cases where there is significant interstitial lung disease, especially when the disease is mainly in the form of coal macules, and not micro or macro-nodules, or when emphysema is present which may distort the lung parenchyma and results in the appearance on chest film of fewer lesions, or when the effect of coal dust is only to produce COPD. He went on to cite a number of studies in support of his conclusions and to support the premise that coal dust can cause obstructive lung disease.

Dr. Cohen's report next addressed the consulting opinions of Dr. Fino, Dr. Renn, Dr. Altmeyer, and Dr. Tuteur. Dr. Fino found that Claimant was disabled due to smoking induced COPD and asthmatic bronchitis with no evidence of "either clinical or legal" pneumoconiosis. Dr. Renn diagnosed chronic bronchitis due to smoking with an asthmatic component, pulmonary emphysema due to smoking, and a severe bronchoreversible obstructive ventilatory defect, but found a total absence of pneumoconiosis. Dr. Cohen countered by pointing out that Drs. Fino and Renn seem to be ignoring the role of coal mine dust in the development of emphysema and obstructive lung disease. Also, he contested their findings, and those of Dr. Altmeyer, by arguing that the objective evidence does not support a conclusion that Claimant suffers from asthma because there is no history of asthma since childhood, but only a lung disease that became apparent after significant exposure to coal mine dust and tobacco smoke. Secondly, his severe obstructive disease always remained severe, even after application of bronchodilator. The only exception to this, according to Dr. Cohen, was one study that barely achieved ATS criteria to be considered meaningful, and even then, Claimant was still left with a severe impairment. Third, Dr. Cohen argued that asthma cannot be diagnosed based on symptoms alone, or based on the patient's medication, but must have corroborating PFTs. And even though one PFT showed reversibility, at no time did Claimant's obstruction reverse to normal. Finally, concerning Dr. Tuteur's conclusions that Claimant's COPD resulted from childhood pneumonias, allergic asthma, and cigarette smoking, Dr. Cohen reiterated his conclusion that Claimant does not have a history of asthma, but also explained that it was "very very unlikely that one episode of pneumonia would cause COPD that would present only after the patient began coal mining." Concerning Dr. Tutor's assignment of a 50% estimate of the chances that an individual with claimant's risk factors would contract COPD as opposed to non-smoking coal miners who have less than a 1% risk of COPD, Dr. Cohen pointed out that Dr. Tuteur has provided no reference to the medical literature to support his claim. Finally, Dr. Cohen concluded that when Dr. Tuteur stated that CWP causes a restrictive abnormality characterized by reduced total lung capacity, may produce impairment of gas exchange, and noted that the x-ray evidence was negative for pneumoconiosis, that it appeared that he was denying "the well proven role ... of coal mine dust in producing obstructive lung disease."

Dr. Cohen next addressed the issue of disability and detailed the progression of Claimant's condition since his prior claims for benefits. Concerning disability, he opined that Claimant was totally disabled from a pulmonary standpoint, and thus unable to return to any type

of coal mining job, including a general inside laborer or a trackman. He based this on the fact that the most recent FEV1 was only 39% prebronchodilator and 42% post. Also, he looked to the moderate gas exchange abnormalities from the ABG studies. Concerning progression of Claimant's pneumoconiosis, Dr. Cohen concluded that Claimant's condition has worsened clinically since 1995. In support, he relied on the increase in the number of exacerbations of COPD, and the worsening of Claimant's gas exchange in his baseline pulmonary function on the ABGs performed when he was not having the exacerbation of his lung disease, but was instead at a stable baseline.

Dr. Robert B. Altmeyer, an internist and pulmonologist, examined the Claimant on August 15, 2001, completing his report on September 18, 2001. (DX 24-25). Dr. Altmeyer considered symptomatology (cough, phlegm production, hemoptysis, wheezing, chest pain with exertion, shortness of breath), employment history (12 years coal mine employment, ending in 1987, as a general laborer and trackman, lifting 75 pounds several times per day), individual history (stroke, diabetes mellitus and hypercholesterolemia, and seizure), family history (noncontributory for lung disease), smoking history (from 1953 until 1978 he smoked 1-2 packs of cigarettes per day), physical examination (breath sounds are severely decreased, mild crackles at the bases which improve but do not completely clear after coughing and deep breathing, and mild wheezing on forced exhalation), chest x-ray (0/1), PFT (severe airway obstruction with a marked degree of trapping), ABG (mild hypoxemia), and an EKG scan (regular). He also reviewed the records of Dr. Knight's March 26, 1991 examination, Dr. Cole's interpretation of a March 26, 1991 x-ray, Dale Boyse's reinterpretation of the March 26, 2001 chest x-ray, Dr. Gaziano's interpretation of an October 13, 1994 chest x-ray, and Dr. Cohen's complete pulmonary evaluation conducted on January 9, 2001. Based on his examination findings and the review of additional medical reports, Dr. Altmeyer concluded that Claimant does not have pneumoconiosis, but instead suffers from naturally occurring asthma, and possibly has some component of airways obstruction due to long-term prior cigarette smoking. He based this diagnosis on the fact that the pattern of the PFT with severe airway obstruction, acute bronchoreversibility and a normal specific diffusing capacity is consistent with naturally occurring asthma. Additionally, the chest x-ray shows absolutely no changes of silicosis or CWP. Also, the finding on physical examination of severely decreased breath sounds, crackles, and mild wheezes are not found in simple CWP or silicosis. Thus, Dr. Altmeyer concluded that his objective findings were all incompatible with CWP, silicosis, or any other occupationally related lung disease. Finally, Dr. Altmeyer concluded that from a pulmonary standpoint, Claimant would not be able to perform his last job in the coal mine, or a job requiring a similar degree of effort or exertion due to the severe degree of airway obstruction.

Dr. Peter G. Tuteur, an internist and pulmonologist, conducted a review of Claimant's medical records review on September 28, 2001. (DX 21, 25). In this first of two reviews in the record, Dr. Tuteur reviewed the following: Dr. Knight's medical reports from March 26, 1991 and October 13, 1994; Dr. Cohen's medical report dated March 5, 2001; Dr. Altmeyer's medical report dated September 18, 2001; hospital records associated with admissions to Good Samaritan Medical Center commencing August 14, 1990, October 15, 1990, November 1, 1990, and January 24, 1991; ABG studies dated January 28, 1991, January 31, 1991, and February 4, 1991; PFT data dated March 26, 1991, October 26, 1994, January 9, 2001, and August 15, 2001. He opined that there was absolutely no convincing information to indicate the presence of CWP, or

any other coal dust-induced disease of the lung that was of sufficient severity and profusion to produce clinical symptoms, physical examination abnormalities, impairment of pulmonary function, or abnormalities on radiographic imaging. Dr. Tuteur, however, did state that Claimant had suffered from “industrial bronchitis,” a condition which is not typically associated with any physiologic impairment, and was resolved following discontinuation of exposure to coal dust. Dr. Tuteur credited cigarette smoking, in conjunction with childhood pneumonia and severe aspiration pneumonia in 1991, which lead to adult respiratory distress syndrome (“ARDS”), with causing Claimant’s COPD. Concerning Claimant’s symptoms, he pointed out that while exercise intolerance and breathlessness are clinical features of CWP, they are also consistent with virtually any primary pulmonary or cardiac disorder. Also, cough, expectoration, wheezing, and chest discomfort are not regular symptoms of CWP. Furthermore, Dr. Tuteur believes that the intensity of breath sounds and intermittently nondescript crackles and wheezes are typical of obstructive airway disease, and not CWP. Ultimately, Dr. Tuteur concluded that while Claimant is totally and permanently disabled, his impairment is not caused even in part by his work in the coal mine industry or CWP, but is the result of smoking, childhood pneumonia, and ARDS.

On April 22, 2003, Dr. Tuteur reviewed the following: independent medical review prepared by Dr. Fino dated February 28, 2002; independent medical review prepared by Dr. Renn dated March 15, 2002; outpatient records associated with care provided by Dr. Kalis and colleagues as well as his office records prior to the time that he assumed care from February 21, 1985 to March 2, 2002; outpatient records associated with care provided by Dr. Elston from February 21, 1985 through October 28, 1998; photocopies of hospital records associated with admission to Bethesda Hospital, Zanesville, Ohio, Good Samaritan Medical Center, Zanesville, Ohio, and Genesis Healthcare System, commencing January 3, 2003, December 19, 1983, October 17, 1985, August 5, 1990, September 11, 1991, November 29, 1991, April 9, 1992, July 14, 1992, October 7, 1993, February 13, 1994, August 2, 1994, January 6, 1995, September 3, 1995, January 6, 1995, September 3, 1995, September 28, 2005, November 6, 1995, March 12, 1996, March 19, 1996, September 11, 1996, and February 26, 2001; PFT data associated with studies dated December 30, 1996, May 11, 1995, and November 19, 2002; ABG studies dated October 15, 1990, two dated November 1, 1990, January 25, 1991, July 16, 1991, September 11, 1991, March 29, 1992, March 1, 1992, April 9, 1992, October 6, 1993, November 4, 1994, February 13, 1994, August 2, 1994, January 9, 1995, March 13, 1995, July 9, 1995, September 2, 1995, September 28, 1995, October 3, 1995, October 21, 1995, November 6, 1995, February 22, 1996, February 23, 1996, September 11, 1996, November 8, 1996, February 26, 2001, and March 26, 2001; 103 chest radiographic reports prepared by 23 different reviewers concerning examinations performed on 64 different dates; and, a report of CT scan of the thorax dated March 14, 2002 that was interpreted by Dr. Belk. (EX 20). Upon summarizing this medical information and concluding that Claimant does suffer from an obstructive pulmonary disease, Dr. Tuteur concluded that based on the totality of available data, there was no convincing information whatsoever to indicate the presence of coal mine dust-induced pulmonary problems. Instead, he opined that Claimant’s COPD was the result of childhood pneumonia, evidence of hay fever, and chronic cigarette smoking as an adult. Dr. Tuteur stated that due to these factors, Claimant was at “possibly even greater than 50%” risk of developing COPD as an adult. On the other hand, had he been a non-smoking coal miner, his chances were “certainly less than 1%” of contracting pneumoconiosis.

Looking at the physical examination, PFT, ABG, CT scan and chest x-ray results, Dr. Tuteur opined that Claimant does not suffer from pneumoconiosis. First, while physical examinations have shown that Claimant has had clear-cut evidence of airflow obstruction in the form of rhonchi, wheezes, crackles, and diminished intensity and prolonged time of expiratory phase, Dr. Tuteur believed that this was typical of a person with COPD due at least in part on cigarette smoke. In contrast, he opined that when coal workers' pneumoconiosis is sufficiently advanced to produce abnormal physical examination, he would expect to find decreased lung expansion, decreased lung size, and persistent late inspiratory crackling sounds, which have not been found in Claimant's case. Second, concerning the PFT results in this case, Dr. Tuteur explained that the results show a slowly progressive severe obstructive ventilatory defect and hyperinflation which he claims are typical for smoke-induced COPD. But if Claimant was suffering from pneumoconiosis, Dr. Tuteur said that he would expect a restrictive abnormality characterized by a reduced total lung capacity instead of the obstructive abnormality with increased total lung capacity as is reflected in Claimant's results. Third, concerning the ABG results, Dr. Tuteur stated that they show some mild impairment of gas exchange which is in essence stable over the years. He opined that while pneumoconiosis may produce impairment of gas exchange, these findings are also typical of cigarette smoke-induced COPD. Fourth, concerning the CT scan and chest x-rays, Dr. Tuteur found that while a number of the x-rays showed emphysema, over 100 of the chest x-rays and the CT scan failed to find changes consistent with pneumoconiosis.

Finally, concerning total disability, Dr. Tuteur considered the totality of medical evidence considered in both his September 28, 2001 report and his April 22, 2003 report, and concluded that Claimant's COPD, caused by childhood pneumonia, allergies, and chronic cigarette smoking, and aggravated by aspiration pneumonia and the development of ARDS, while permanently and totally disabling, is not due, even in part to pneumoconiosis, the inhalation of coal dust, or the development of coal mine dust related pulmonary problems of any sort. He concluded that had Claimant never worked in the coal mine industry, his clinical course and course and type and severity of his current health problems would have been no different than depicted in the records.

Dr. Tuteur was deposed by the Employer on February 3, 2004. (EX 26). While the majority of his deposition repeated the findings of his earlier written report, there were a few important additions. During direct examination, Dr. Tuteur stated that there is a possibility that coal mine dust exposure contributed to Claimant's air flow obstruction, but considering the other risk factors, with reasonable medical certainty, he opined that exposure did not contribute in this case. (EX 26: 22-23). During cross-examination Dr. Tuteur admitted that the risk factors he had focused on were childhood pneumonia, hay fever, cigarette smoking, and aspiration pneumonia resulting in esophageal reflux disease. (EX 26: 43). He was then asked what the clinical record included concerning Claimant's childhood pneumonia. (EX 26: 43). In response he said, "Little. All it tells us that I could find is that there was a notation that he had pneumonia as a child." He then launched into an explanation of why childhood pneumonia is a risk factor for developing obstructive lung disease. (EX 26: 43-44). When the examiner redirected Dr. Tuteur to the extent of the documentation of Claimant's childhood pneumonia, the doctor admitted that the clinical data does not say anything about the extent or severity of this pneumonia. (EX 26:44-45). Next, Dr. Tuteur was asked about the clinical evidence to support esophageal reflux disease, and

responded that the record showed that the disease was suspected but never confirmed by any test. (EX 26: 57). The rest of the deposition focused mainly on explaining and defending the studies Dr. Tuteur utilized to support his interpretation of the medical data, and explaining the problems with studies that disagreed with his analysis and those relied upon by Dr. Cohen and to some extent Dr. Parker. (EX 26:30-31, 60-88; 30).

Dr. Gregory L. Fino, an internist, pulmonologist, and B-reader, submitted a medical evidence review report on February 28, 2002. (EX 27). His report included an analysis of the following records: Office records from February 21, 1985 to October 28, 1998; hospital admission records from September 11, 1991 through September 16, 1991, November 1, 1990 through November 3, 1990, January 24, 1991 through February 4, 1991, July 14, 1992; October 19, 1993 through October 29, 1993, February 13, 1994 through February 15, 1994; August 2, 1994 through August 5, 1994, September 11, 1996 through September 17, 1996, February 26, 2001 through March 4, 2001; emergency room records dated February 19, 1992, November 4, 1993, July 15, 1994, October 26, 1994, March 18, 1998, August 2, 1998, July 8, 2001; DOL examination records dated March 13, 1991, October 13, 1994, January 9, 2001; Medical examination records by Dr. Altmeyer dated September 18, 2001; medical record review by Dr. Tuteur dated September 28, 2001; chest x-ray film dated January 9, 2001; chest x-ray interpretations dated February 25, 1985, October 14, 1985, March 13, 1991, April 2, 1992, September 15, 1993, January 6, 1995, February 15, 1995, April 11, 1995, July 9, 1995, September 28, 1995, March 10, 1995, March 12, 1996, November 18, 1996, May 18, 1997, August 5, 1998, December 27, 1998, January 1, 2001; PFTs dated December 30, 1986, May 11, 1995; and ABGs dated April 9, 1992, October 6, 1993, July 16, 1991. Dr. Fino diagnosed smoking induced COPD and asthmatic bronchitis. He also stated that he did not find objective medical evidence of either CWP or legal CWP. He concluded that Claimant was disabled due to lung disease, but the inhalation of coal dust played no role in the disability.

Dr. Fino was deposed on February 24, 2004. (EX 27). Prior to the deposition, Employer had forwarded him Director's exhibits 1-34, Claimant's exhibits 1-4, and Employer's exhibits 1-25 for review. (EX 27: 6). Dr. Fino stated that these additional records did not change any of the opinions from his February 28, 2002 report. He did testify, however, that the serial ABG studies he reviewed show some hypoxemia as of 2001. (EX 27: 15). Also, he stated that "[o]verall, [Claimant's] pulmonary capacity function has worsened in the last ten years." Next, when questioned concerning the etiology of Claimant's condition, Dr. Fino stated that Claimant has sufficient coal dust exposure and cigarette exposure to cause the obstructive abnormality, and that both coal dust and cigarette smoke can cause obstructive abnormalities. (EX 27: 17-18). But, he claimed to be able to distinguish the cause of Claimant's condition based on the medical literature. (EX 27: 18). Furthermore, when asked whether he felt Claimant had had a material change in his physical condition since 1995, Dr. Fino stated that Claimant "is as disabled as he was in the mid-90s but his lung function and his blood gases, in my opinion, have worsened over time." (EX 27: 27). Finally, the remainder of the direct and cross-examination focused on applying the medical literature to this claim in support of his diagnosis. (EX 27: 30).

Dr. Joseph J. Renn, an internist, pulmonologist, and B-reader, performed a review of medical evidence and submitted a report on March 15, 2002. (EX 6, 9). Dr. Renn reviewed the following: Dr. Elston's office records from 1985 through 1988; Good Samaritan medical records

dating from 1990 through 2001; Dr. Knight's independent medical reviews dated 1991 and 1994; Dr. Cohen's independent medical review dated January 9, 2001; Genesis Healthcare medical treatment records from 2001; Dr. Altmeyer's independent medical evaluation dated September 19, 2001 for his examination of August 15, 2001; Dr. Tuteur's independent medical review dated September 28, 2001; 11 electrocardiographs taken between 1986 and 2001; echocardiograms dated February 25, 1985, November 2, 1990, March 25, 1996, and September 11, 1996; PFTs dated December 30, 1986, March 26, 1991, January 9, 2001, and August 15, 2001; ABGs dated March 26, 1991, October 13, 1994, January 9, 2001, and August 8, 2001; 53 chest radiograph interpretations dated between February 25, 1985 and August 15, 2001; holter monitor interpretation dated April 11, 1995; CT scans dated September 5, 1995, March 23, 1996, August 7, 1990, and November 16, 1990; a lung scan interpretation dated November 4, 1993, a whole body white cell study interpretation dated March 18, 1996; a left hip aspiration interpretation dated March 16, 1996; an 8 to 50 pack year smoking history; and Claimant's 12 year work history records. (EX 9). Dr. Renn diagnosed Claimant as suffering from chronic bronchitis with an asthmatic component and pulmonary emphysema due to tobacco smoking. Also, he opined that Claimant suffered from severe to very severe, but significantly bronchoreversible obstructive ventilatory defect. Finally, he concluded that while Claimant does have a total and permanent respiratory impairment, Claimant does not suffer from pneumoconiosis or any other coal mine dust-induced disease process, but his condition is wholly the result of tobacco smoke-induced diseases. Dr. Renn, however, provided no explanation as to why he ruled out coal dust exposure as a cause and accredited Claimant's condition solely to tobacco use.

Dr. Renn was deposed by the Employer on February 26, 2004, after he had the opportunity to review the remaining evidence in the record, and he repeated the findings of his earlier written report. (EX 28). Also, he expanded on why he believed that Claimant's condition was the result of smoking and was not caused by coal dust exposure. (EX 28: 70-71). First, he opined that Claimant has a physiologic pattern that is consistent with tobacco smoking-induced disease with an asthmatic component, and not CWP. (EX 28: 48-49, 71). Second, since Claimant has no radiographic evidence of CWP, he has a greater severity of reduction in his ventilatory function than has been found in any person with CWP. (EX 28: 49, 72). Third, Claimant has hypercarbia, which can never occur in CWP, but it occurs in tobacco smoke-included disease of chronic bronchitis, especially chronic bronchitis with an asthmatic component. (EX 28: 81). Finally, a large portion of the deposition focused on explaining and defending the studies Dr. Renn utilized to support his interpretation of the medical data, and explaining the problems with studies that disagreed with his analysis and those relied upon by Dr. Cohen and to some extent Dr. Parker. (EX 28).

Dr. John E. Parker, an internist, pulmonologist, and B-reader, performed a review of medical evidence and submitted a report on January 6, 2004. (CX 3). First, Dr. Parker reviewed PFT and ABG studies dated December 30, 1986 (PFT only), March 26, 1991, October 13, 1994, January 9, 2001, August 15, 2001, and November 19, 2002 (PFT only), and standardized the predicted normals in order to best view progression over time, using the Intermountain Thoracic criteria. Concerning the PFTs, he concluded that spirometry shows severe obstruction with minimal acute bronchodilator responsiveness. And, even though post bronchodilator values showed improvement, they did not improve even to the moderately impaired range. Also, lung volumes showed hyperinflation and diffusing capacity was moderately reduced. Concerning the

ABG studies, Dr. Parker recognized a pattern of progressive impairment of gas exchange. Second, Dr. Parker reviewed a large number of chest x-ray interpretations from the mid 1980's through 2001, and noted that a large number found evidence of COPD/emphysema. Evaluating the 13 x-rays which were done under the ILO classification system, he concluded that none were positive for opacities consistent with radiographic evidence of clinical pneumoconiosis. Third, Dr. Parker reviewed medical treatment records from Dr. Elston, Dr. Kalis, Dr. Albrini, Dr. Mumma, Bethesda North Hospital, and Good Samaritan Hospital, dating from 1985 through 2003. While he found these records indicated that Claimant suffered from a number of ailments over the years, Dr. Parker emphasized the diagnoses of COPD/emphysema. He explained that the records reveal a long standing-severe COPD/emphysema with symptoms beginning early in Claimant's coal mine years that have become increasingly frequent and severe exacerbations in recent years. Fourth, Dr. Parker reviewed the physical examination reports and non-examination reviews of record, including reports by: Dr. Knight, Dr. Cohen, Dr. Altmeyer, Dr. Tuteur, Dr. Fino, and Dr. Renn. He noted that all of these physicians, with exception of Dr. Tuteur, specified that Claimant's chronic respiratory condition was by itself disabling. Dr. Tuteur, however, emphasized the combination of respiratory and other impairments as disabling. Finally, Dr. Parker stated that the various examinations reports and treating records show numerous physical findings consistent with chronic lung disease at various times, such as rales, rhonchi, crackles, diminished breath sounds, and wheezing.

Considering all of the evidence, Dr. Parker concluded that Claimant suffers from pneumoconiosis, manifested as a severe obstructive lung disease, including severely reduced FEV1 and FEV1/FVC ratios, reduced diffusing capacity, and progressively worsening hypoxemia, with normal or hyperinflated lung volumes. Also, despite the fact that the chest x-rays that were interpreted negative for clinical pneumoconiosis, Dr. Parker believes that the epidemiological evidence for a casual link between coal dust and COPD is, in this case, "massive and irrefutable." Dr. Parker cited a number of scientific references to support these conclusions.

Concerning etiology, Dr. Parker opined that Claimant's COPD was caused in substantial part by both his 12 years of coal mine employment, ending in 1987, and his approximately 30 pack-year smoking history ending around 1980. Also, while he acknowledged that prolonged tobacco abuse causes COPD, he also noted that 12 years of underground exposure is also sufficient to cause COPD. Furthermore, he pointed out that Claimant stopped smoking 5-7 years prior to the end of his coal mine work, and that Claimant began to become symptomatic early in his coal mining career, and that by 1986, 6-8 years after he quit smoking and one year prior to leaving the mines, his FEV 1 had become severely reduced. Based on the scientific evidence regarding the etiology of COPD, Claimant's history, physical findings, symptoms, PFT, ABG and x-ray evidence, Dr. Parker concluded that both smoking and coal mine employment had contributed to Claimant's COPD.

Concerning changes in Claimant's condition since his 1991 and 1995 claims, Dr. Parker explained that the numerous ABG studies show clear worsening, manifested by falling pO₂ as of 2001 compared to results in 1994 and 1991. Also, he noted that the treatment records show progressive worsening and frequency of exacerbations since the mid 1990's. As a result, Dr. Parker concluded that Claimant's condition has worsened.

Concerning total disability, Dr. Parker expressed no doubt in his assertion that Claimant was totally unable from a respiratory perspective to perform the tasks of either a general inside laborer or a trackman. In support he noted the severity of the reductions in FEV 1 and diffusing capacity, as well as Claimant's symptoms and complaints.

Finally, Dr. Parker addressed the opinions of Drs. Renn, Fino, Tuteur, and Altmeyer, who found that coal dust did not contribute to Claimant's condition. First, Dr. Altmeyer, Dr. Renn, and Dr. Fino all concluded that asthma was a possible cause of Claimant's condition. In response, Dr. Parker opined that Claimant does not have asthma, and even if he does, it is not a substantial cause of the respiratory disability. He based this conclusion on the fact that Claimant's "response to bronchodilators only barely reached a level of statistical significance (and not on every occasion) and that administering of the bronchodilators never increased his FEV1 even to the level of moderate disease, let alone to the complete reversibility that is common with asthma." Furthermore, Dr. Parker maintains that the "persistent presence of severely reduced FEV1 and FEV1/FVC ratios after bronchodilatory is strong evidence for the primacy of COPD, most likely emphysema and chronic bronchitis in causing [Claimant's] respiratory impairment." Finally, Dr. Parker addressed Dr. Tuteur's conclusions that excluded coal dust as a cause of Claimant's COPD in favor of cigarette smoking. Dr. Parker stated that Dr. Tuteur's opinion (smoking, childhood pneumonia, and allergic diathesis created a 50% risk of COPD, compared to a less than 1% risk among coal miners) is not supported by the large body of scientific literature in the NIOSH 1995 Criteria Document.

Treatment Records

Over 1,700 pages of medical treatment records have been submitted in conjunction with this claim; 1,654 by the Employer and 60 by the Claimant. These records include treating physician histories: Dr. Storm Elston from February 1985 through November 1998 (EX 13); Dr. Perry Kalis from November 1998 through March 2002 (EX 12, 19); Dr. Albrini from March 2002 through November 2002 (EX 22); and Dr. Mumma from April 2001 through January 2004 (EX 18, 23). Also submitted are 20 years of hospital treatment records spanning December 1983 through December 2003. All of these records are covered under the Genesis Healthcare System, and include the Good Samaritan Medical Center and Rehabilitation center, and Bethesda Hospital. (EX 14, 17, 24, 25; DX 27; CX 1).¹²

Smoking History

At the hearing, Claimant testified that he smoked a pack of cigarettes a day from 1952 to 1975, and from 1975 to 1978, he smoked ½ packs per day. (Tr. 33-34). He stated that he completely quit smoking in 1978, and remains a non-smoker. (Tr. 34). Claimant's testimony is consistent with his responses to Employer's interrogatories. (DX 22). Also, Mrs. Balsley testified that since she met Claimant in 1987, she has never seen him smoke. (Tr. 45-46). Dr.

¹² Since the treating physician exhibits and hospital treatment records overlap, with some reports appearing in two or three different exhibits, the undersigned has taken the Claimant's lead, and has listed the applicable reports in chronological order, starting with the December 1983 admission report through Dr. Mumma's December 2003 examination. Also, parallel citations to the record are not included. The complete chronological summary of those notes appear as Appendix A to this Decision and Order. While Claimant's brief contains a similar chronological summary, which has been a helpful aid, the attached summary constitutes an independent analysis of these notes.

Cohen reported that Claimant smoked cigarettes from 1953 to 1979 at a rate of 1 pack per day for 23 years and ½ packs per day for 3 years. (DX 10). Dr. Altmeyer reported that Claimant smoked from 1953 until 1978 at a rate of 1-2 packs of cigarettes per day (DX 23). There were also hundreds of varying references to smoking history in the medical treatment records. (DX 27; CX 1; EX 12, 14, 17-19, 23-25, 27).¹³ Claimant's testimony is consistent with his answer to the interrogatories, Ms. Balsley's testimony, and Dr. Cohen's report. Only Dr. Altmeyer's report diverges from Claimant's testimony, and then only in relation to the rate of smoking and not the duration. As a result, I find Claimant's testimony credible. Therefore, I find that Claimant smoked from 1952 to 1975 at a rate of one pack per day, and 1976 through 1978 at a rate of ½ packs per day, equaling 24.5 pack years of cigarette smoking. Also, Claimant quit smoking in 1978.

PRIOR SUBMITTED MEDICAL EVIDENCE

X-RAY REPORTS

Exhibit	Date of X-ray	Date of Reading	Physician/Qualifications	Interpretation
DX 31	03/26/91	03/28/91	Boyse ¹⁴	Negative
DX 31	03/26/91	04/10/91	Cole, B-reader	Negative
DX 32	10/13/94	10/31/94	Gaziano, B-reader	0/1 tt
DX 32	10/13/94	10/17/94	Boyse	Negative

PULMONARY FUNCTION STUDIES

Exhibit/ Date	Co-op./ Undst./ Tracings	Age/ Height¹⁵	FEV₁	FVC	MVV	FEV₁/ FVC	Qualifying Results
DX 31 3/26/91	Good/ Good/ Yes	53 70"	1.47	2.96	86	50	Yes

¹³ While Employer, in its brief, makes reference to the fact that evidence within the medical treatment records supports a conclusion of a greater than 25 pack year history, it cites no specific exhibits or page numbers. Also, since these records were created for the purpose of treating Claimant's condition, and not for the purpose of black lung litigation, I accord them less weight than the reports created by examining physicians in preparation for the instant claim.

¹⁴ Dr. Boyse has been a certified A-reader according to the June 17 2004 B-reader list since December 14, 1970.

¹⁵ In footnote 9, above, Claimant's height was determined to be 71 inches.

DX 32 10/13/94	Good/ Good/ Yes	57 72"	1.14	2.63	35	43	Yes
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All values are pre-bronchodilator

ARTERIAL BLOOD GAS STUDIES

Exhibit	Date	pCO ₂	pO ₂	Qualifying
DX 31	03/26/91	40.6	74.9	No
DX 32	10/13/94	41.5	74.5	No

All values are pre-exercise

Narrative Medical Evidence

Dr. Paul Knight examined the Claimant on March 26, 1991. (DX 31). Based on symptomatology (sputum, wheezing, dyspnea, cough, orthopnea, and chest pain), employment history (15 years as a general inside laborer), individual history (wheezing, chronic bronchitis, bronchial asthma, high blood pressure, 1991 hip fracture, and a stroke in 1990), family history (diabetes), smoking history (26 pack-pack year history), physical examination (percussion is clear with no wheezes or rales, but distant breath sounds), chest x-ray (clear), PFT (moderate obstructive ventilatory defect), ABG (non-qualifying), and an EKG (normal sinus rhythm), Dr. Knight diagnosed a moderate respiratory impairment due to chronic bronchitis. He stated that due to stroke in 1990 and the left hip fracture in 1991, Claimant has left side hemiplegia, and was unable to exercise due to being wheelchair bound. He opined that Claimant's moderate impairment due to chronic bronchitis, which was primarily caused by cigarette smoking with a secondary aggravating factor of coal dust exposure, would likely be sufficient to keep him from performing his last coal mining job. But, even without the respiratory impairment, Claimant's other physical ailments are by themselves completely disabling.

Dr. Knight examined the Claimant a second time on October 13, 1994. (DX 32). Based on symptomatology (sputum, wheezing, dyspnea, orthopnea, paroxysmal nocturnal dyspnea), employment history (14 years coal mine employment as a general inside laborer and a trackman), individual history (childhood pneumonia, wheezing, chronic bronchitis, bronchial asthma, high blood pressure, partial left-side paralysis due to 1990 stroke, pneumonia in 1994, 1991 hip fracture, 1994 total hip replacement), family history (heart disease), smoking history (16 years at ½ pack per day), physical examination (percussion is clear with no evident wheezing, but has distant breath sounds), chest x-ray (clear), PFT (moderate obstructive ventilatory defect with moderate restriction present that cannot be further characterized without lung volumes), ABG (non-qualifying), and an EKG (normal sinus rhythm, non-specific ST and T change), Dr. Knight diagnosed moderate COPD and chronic bronchitis due to a combination of previous dust exposure in the course of his coal mining work as well as on previous tobacco usage. He opined that Claimant's respiratory impairment is disabling enough to prevent him from doing coal mining type work, but he is also disabled as a result of his CVA.

Treatment Records

The treatment records submitted in conjunction with the prior claim is largely a repeat of Dr. Elston's treatment notes reviewed in App. A. (EX 17). The six entries below constitute the only addition to the newly submitted medical treatment evidence.

11/1/90 – X-ray reading by Dr. Boyse – Lungs: clear. (DX 32).

2/5/91 – Discharge summary by Dr. Krohn – During the induction of the anesthetic, the patient vomited and was sucked out, no actual fluid was sucked out of the trachea at the time, and I could not hear any fluid in the lungs on auscultation but postoperatively, within an hour after arriving in recovery room, the patient gradually became more short of breath, asthmatic, and was seen by Dr. Scheerer who inserted an endotracheal tube. X-rays taken the evening of surgery showed increasing density in the left base, diaphragm elevation, and infiltrate developing at the left base. On the 26th, another portable x-ray showed a left lower lobe atelectasis since the previous exam. By the 27th, the film showed improvement and chest x-ray was read as normal. The last x-ray of the chest taken on the 31st of January showed clearing of the previously described infiltrate with blunting of the right left costophrenic angle, unchanged. (DX 32).

4/23/91 – Examination by Dr. Elston – Lungs: clear. (DX 32).

9/11/91 – Examination by Dr. Short – 1+ coarse wheezes throughout with a rare crackle in the left base, but no pleural rubs or no CVA tenderness. X-ray showed possible lower left lobe infiltrate. Impression: COPD exacerbation secondary to probable pneumonia. (DX 32).

7/14/92 – Examination by Dr. Elston – Decreased breath sounds and scattered rhonchi. Impression: COPD. (DX 32).

9/24/93 – Examination by Dr. Elston – Decreased breath sounds but clear. (DX 32).

DISCUSSION AND APPLICABLE LAW

Mr. Balsley's claim was made after March 31, 1980, the effective date of Part 718, and must therefore be adjudicated under those regulations. To establish entitlement to benefits under Part 718, Claimant must establish, by a preponderance of the evidence, the following elements:

1. That he suffers from pneumoconiosis;
2. That the pneumoconiosis arose, at least in part, out of coal mine employment;
3. That the claimant is totally disabled; and
4. That the total disability is caused by pneumoconiosis.

See §§ 719.3, 718.202, 718.203, 718.204; *Gee v. W.G. Moore*, 9 B.L.R. 1-4, 1-5 (1986); *Roberts v. Bethlehem Mines Corp.*, 8 B.L.R. 1-211, 1-212 (1985). Failure to establish any of these elements precludes entitlement. *Anderson v. Valley Camp of Utah, Inc.*, 12 B.L.R. 1-111, 1-112 (1989); *Trent v. Director, OWCP*, 11 B.L.R. 1-26, 1-27 (1987).

Subsequent Claim

Both Claimant's 1991 and 1994 applications for benefits were denied more than one year prior to the filing of his third claim in November of 2000. The provisions of § 725.309 apply to new claims that are filed more than one year after a prior denial. Section 725.309 is intended to provide claimants relief from the ordinary principles of *res judicata*, based on the premise that pneumoconiosis is a progressive and irreversible disease. See *Lukman v. Director, OWCP*, 896 F.2d 1248 (10th Cir. 1990); *Orange v. Island Creek Coal Company*, 786 F.2d 724, 727 (6th Cir. 1986); § 718.201(c) (Dec. 20, 2000). Section 725.309(d) provides that:

If the earlier miner's claim has been finally denied, the later claim shall also be denied, on the grounds of the prior denial, unless the deputy commissioner determines that there has been a material change in conditions or the later claim is a request for modification and the requirements of § 725.310 are met.

The Benefits Review Board defined "material change in conditions" under § 725.309(d) as occurring when a claimant establishes, by a preponderance of the evidence developed subsequent to the prior denial, at least one of the elements of entitlement previously adjudicated against the claimant. See *Allen v. Mead Corp.*, 22 B.L.R. 1-61 (2000). The Board has also held that a material change in conditions may only be based upon an element which was previously denied. *Caudill v. Arch of Kentucky, Inc.*, 22 B.L.R. 1-97 (2000) (en banc on recon.) (where Administrative Law Judge found that claimant did not establish pneumoconiosis and did not specifically address total disability, the issue of total disability may not be considered in determining whether the newly submitted evidence is sufficient to establish a material change in conditions). Lay testimony alone is insufficient to establish a material change in conditions. *Madden v. Gopher Mining Co.*, 21 B.L.R. 1-122 (1999).

In *Tennessee Consolidated Coal Co. v. Director, OWCP [Kirk]*, 264 F.3d 602 (6th Cir. 2001), the Sixth Circuit held that, under *Sharondale Corp. v. Ross*, 42 F.3d 993 (6th Cir. 1994), it is insufficient for the ALJ to merely analyze the newly submitted evidence to determine whether an element previously adjudicated against the claimant has been established. An administrative law judge must also compare the sum of the newly submitted evidence against the sum of the previously submitted evidence to determine whether the new evidence is substantially more supportive of claimant. *Kirk*, 264 F.3d at 609. However, when comparing the newly submitted evidence against the previously submitted evidence, only a substantial difference in the bodies of evidence is required, not a complete absence of evidence at the earlier time. *Id.* at 610. It is legal error for an administrative law judge not to show that there was a worsening of Claimant's condition on the element selected to show a material change. *Id.* at 609.

Claimant's 1994 claim was denied because he failed to establish pneumoconiosis as defined by the Act, that his pneumoconiosis was caused by coal mine employment, and because

he failed to establish that his total disability was caused by pneumoconiosis. (DX 32). The Director, however, found that the evidence established the Claimant's total disability. (DX 32). Claimant's subsequent claim must be denied on the basis of the prior denial, unless he establishes that he suffers from pneumoconiosis or that his total disability is caused by pneumoconiosis. For purposes of duplicate claim analysis only, it must be assumed that Claimant has established that he is totally disabled. If Claimant establishes a material change in conditions in one of the elements previously adjudicated against him, the undersigned will conduct a *de novo* review of all of the evidence of record, then Claimant must establish by a preponderance of the evidence that he is totally disabled.

Pneumoconiosis

In establishing entitlement to benefits, Claimant must initially prove the existence of pneumoconiosis under § 718.202. Claimant has the burden of proving the existence of pneumoconiosis, as well as every element of entitlement, by a preponderance of the evidence. See *Director, OWCP v. Greenwich Collieries*, 512 U.S. 267 (1994). Pneumoconiosis is defined by the regulations:

(a) For the purpose of the Act, "pneumoconiosis" means a chronic dust disease of the lung and its sequelae, including respiratory and pulmonary impairments, arising out of coal mine employment. This definition includes both medical, or "clinical" pneumoconiosis and statutory, or "legal" pneumoconiosis.

(1) *Clinical Pneumoconiosis*. "Clinical pneumoconiosis" consists of those diseases recognized by the medical community as pneumoconiosis, i.e., conditions characterized by permanent deposition of substantial amounts of particulate matter in the lungs and the fibrotic reaction of the lung tissue to that deposition caused by dust exposure in coal mine employment. This definition includes, but is not limited to, coal workers' pneumoconiosis, anthracosilicosis, anthracosis, anthrosilicosis, massive pulmonary fibrosis, silicosis or silicotuberculosis, arising out of coal mine employment.

(2) *Legal Pneumoconiosis*. "Legal pneumoconiosis" includes any chronic lung disease or impairment and its sequelae arising out of coal mine employment. This definition includes, but is not limited to, any chronic restrictive or obstructive pulmonary disease arising out of coal mine employment

(b) For the purposes of this section, a disease "arising out of coal mine employment" includes any chronic pulmonary disease or respiratory or pulmonary impairment significantly related to, or substantially aggravated by, dust exposure in coal mine employment.

(c) For purposes of this definition, "pneumoconiosis" is recognized as a latent and progressive disease which may first become detectable only after the cessation of coal mine dust exposure.

Sections 718.201(a-c).

Section 718.202(a) sets forth four methods for determining the existence of pneumoconiosis.

(1) Under § 718.202(a)(1), a finding that pneumoconiosis exists may be based upon x-ray evidence. Also, if the film quality is poor or unreadable, then the study may be given little or no probative value as it is very poor quality. *Gober v. Reading Anthracite Co.*, 12 B.L.R. 1-67 (1988).

The newly submitted evidence contains 14 interpretations of four chest x-rays. All of the readings, with exception of Dr. Altmeyer's interpretation of the August 15, 2001 film were negative for pneumoconiosis. While Dr. Altmeyer did not conclude the August 15, 2001 film was negative for the disease; he only gave it a 0/1 rating which does not qualify under §718.102(b). As a result, I find that the preponderance of the newly submitted chest x-ray evidence fails to establish pneumoconiosis.¹⁶

The chest x-ray evidence submitted in conjunction with the 1991 and 1994 claims was also either negative or non-qualifying. As a result, the newly submitted x-ray evidence as compared to the sum of the previously submitted evidence fails to demonstrate a substantial change in Claimants condition as reflected in chest x-ray evidence. This is because none of the x-rays in evidence, old or new, show qualifying values as required by §718.102(b). Therefore, I find that Claimant has failed to establish a material change in condition due to the presence of pneumoconiosis under subsection (a)(1).

(2) Under § 718.202(a)(2), a determination that pneumoconiosis is present may be based, in the case of a living miner, upon biopsy evidence. The evidentiary record does not contain any biopsy evidence. Therefore, I find that the Claimant has failed to establish the existence of pneumoconiosis through biopsy evidence under subsection (a)(2).

(3) Section 718.202(a)(3) provides that pneumoconiosis may be established if any one of several cited presumptions are found to be applicable. In this case, the presumption of § 718.304 does not apply because there is no evidence in the record of complicated pneumoconiosis. Section 718.305 is not applicable to claims filed after January 1, 1982. Finally, the presumption of § 718.306 is applicable only in a survivor's claim filed prior to June 30, 1982. Therefore, Claimant cannot establish pneumoconiosis under subsection (a)(3).

¹⁶ There is a large number of x-ray interpretations included as part of Claimant's treatment history. There is no evidence in the record as to the x-ray reading credentials of these physicians. Also, these interpretations were all related to the treatment of Claimant's condition, and not for the purpose of determining the existence or extent of pneumoconiosis. Finally, there is no record of the film quality for any of these x-rays. As a result, despite the fact that the records unanimously suggest no pneumoconiosis, the x-ray results are not in compliance with the quality standards of §718.102 and Appendix A to Part 718. Therefore, I accord the x-ray interpretations contained in the treatment records no weight for the purpose of determining whether Claimant suffers from pneumoconiosis under § 718.202(a)(1).

(4) The fourth and final way in which it is possible to establish the existence of pneumoconiosis under § 718.202 are set forth in subsection (a)(4) which provides in pertinent part:

A determination of the existence of pneumoconiosis may also be made if a physician, exercising sound medical judgment, notwithstanding a negative x-ray, finds that the miner suffers or suffered from pneumoconiosis as defined in § 718.201. Any such finding shall be based on electrocardiograms, pulmonary function studies, physical performance tests, physical examination, and medical and work histories. Such a finding shall be supported by a reasoned medical opinion.

§ 718.202(a)(4).

This section requires a weighing of all relevant medical evidence to ascertain whether or not the claimant has established the presence of pneumoconiosis by a preponderance of the evidence. Any finding of pneumoconiosis under § 718.202(a)(4) must be based upon objective medical evidence and also be supported by a reasoned medical opinion. A reasoned opinion is one which contains underlying documentation adequate to support the physician's conclusions. *Fields v. Island Creek Coal Co.*, 10 B.L.R. 1-19, 1-22 (1987). Proper documentation exists where the physician sets forth the clinical findings, observations, facts, and other data on which he bases his diagnosis. *Oggero v. Director, OWCP*, 7 B.L.R. 1-860 (1985). A brief and conclusory medical report which lacks supporting evidence may be discredited. *See Lucostic v. United States Steel Corp.*, 8 B.L.R. 1-46 (1985); *see also, Mosely v. Peabody Coal Co.*, 769 F.2d 257 (6th Cir. 1985). Further, a medical report may be rejected as unreasonable where the physician fails to explain how his findings support his diagnosis. *See Oggero*, 7 B.L.R. 1-860.

The newly submitted evidentiary record contains one narrative medical opinion by a treating physician and four reports by examining physicians. The record also contains six medical records reviews by non-examining physicians, one of which was also an examining physician who submitted a subsequent medical evidence review. In total, the newly submitted evidentiary record includes narrative medical opinions by seven different physicians.

First, concerning treating physicians, "[T]he opinions of treating physicians are not necessarily entitled to greater weight than those of non-treating physicians in black lung litigation." *Eastover Mining Co. v. Williams*, 338 F.3d 501 (6th Cir. 2003). "[I]n black lung litigation, the opinions of treating physicians get the deference they deserve based on their power to persuade." *Id.* at 510; 20 C.F.R. § 718.104(d). "A highly qualified treating physician who has lengthy experience with a miner may deserve tremendous deference, whereas a treating physician without the right pulmonary certifications should have his opinion appropriately discounted." *Id.* In addition, appropriate weight should be given as to whether the treating physician's report is well-reasoned and well-documented. *See Peabody Coal Co. v. Groves*, 277 F.3d 829 (6th Cir. 2002); *McClendon v. Drummond Coal Co.*, 12 B.L.R. 2-108 (11th Cir. 1988). Finally, under the 2001 amended regulations, an administrative law judge may rely upon the well-reasoned and well-documented opinion of a treating physician as substantial evidence in awarding that physician's opinion controlling weight based upon four factors: (1) nature of

relationship; (2) duration of relationship; (3) frequency of treatment; and (4) extent of treatment. § 718.104(d) (2002). While this claim does not fall under the new regulations since it was filed prior to their applicability, I find that these factors are helpful in determining the weight to apply to a treating physician's medical opinion.

Next, concerning examining physicians as opposed to those issuing reports upon completion of a medical records review, a non-examining physician's opinion may constitute substantial evidence if it is corroborated by the opinion of an examining physician or by the evidence considered as a whole. *Newland v. Consolidation Coal Co.*, 6B.L.R. 1-21286 (1984).

Dr. Mumma, Claimant's most recent treating physician, submitted a narrative medical report. (CX 4). Based on an accurate employment and smoking history; clinical examination (abnormal); bronchoscopies, CT scans, and chest x-rays (severe obstructive lung disease); PFTs (severe impairment); and ABG studies (chronic hypoxemia when he becomes sick), Dr. Mumma diagnosed CWP with secondary pulmonary fibrosis. While he considered that there may be a casual link between tobacco use and obstructive lung disease, Dr. Mumma believes the severity and progression of Claimant's obstructive lung disease is more typical of CWP with fibrosis. Dr. Mumma set forth clinical observations and findings, and his reasoning is supported by adequate data. His opinion is well-reasoned and well-documented. Therefore, despite the fact that Dr. Mumma is neither an internist nor pulmonologist, I find that his opinion is entitled to great probative weight based on his status as Claimant's most recent treating physician.

Considering Dr. Mumma's opinion and supporting treatment notes in relation to the four factors of § 718.104(d) (2002), I find the following:

- 1) Nature of relationship – Dr. Mumma has treated Claimant repeatedly for both his respiratory and pulmonary condition, as well as other ailments (App. A);
- 2) Duration of relationship – Dr. Mumma stated in his medical narrative report that he had been treating Claimant for approximately 18 months for severe COPD (CX 6; App. A);
- 3) Frequency of treatment – The medical treatment records reflect that Dr. Mumma examined Claimant on 15 occasions between March 13, 2002 and December 3, 2003 concerning his cardiopulmonary condition (EX 23-25; CX 1; App. A); and
- 4) Extent of treatment – Dr. Mumma has treated Claimant over this period with a regimen of medications, by ordering additional testing, including chest x-rays and ABG studies, and by requesting consulting opinions from additional doctors. (EX 23-25; CX 1; App. A).

Dr. Cohen, an internist, pulmonologist, and B-reader, submitted both an examination report and a subsequent medical evidence review. (CX 2; DX 8, 10). Dr. Cohen found that Claimant was suffering from COPD caused by cigarette smoking and coal dust exposure, severe

obstructive lung disease based on his PFT with only slight improvement with bronchodilators. In his subsequent report Dr. Cohen found that the examination reports were consistent with chronic lung disease, the PFTs demonstrated severe obstructive lung disease that has deteriorated over the years since he stopped mining and smoking, ABG studies that reflect mild hypoxemia at rest which progressed to moderate hypoxemia by August 2001. Dr. Cohen also considered an adequate account of Claimant's smoking and coal mine employment histories, finding it significant that he quit smoking 5-7 years prior to cessation of coal mining. Dr. Cohen has relied on objective observations and findings, and his reasoning is supported by adequate data. His opinion is well-reasoned and well-documented. Noting his use of supporting medical literature, I conclude that Dr. Cohen's opinion is entitled to great probative weight based on the fact that he personally examined the Claimant, and enhanced by his credentials as a board-certified internist and pulmonologist.

Dr. Altmeyer, an internist and pulmonologist, submitted an examination report. (EX 22). He relied upon objective test results and prior findings by other physicians to conclude that Claimant does not have pneumoconiosis, but instead suffers from naturally occurring asthma, and possibly some component of airways obstruction due to long-term cigarette smoking. He explained that the PFT results were consistent with naturally occurring asthma. In his opinion, the x-ray shows no changes of silicosis or CWP, and the findings from the physical examination are not found in simple CWP or silicosis. Dr. Altmeyer has relied on objective observations and findings, and his reasoning is supported by adequate data. His opinion is well-reasoned and well-documented. I find that Dr. Altmeyer's opinion is entitled to great probative weight based on the fact that he personally examined the Claimant, and enhanced by his credentials as a board-certified internist and pulmonologist.

Dr. Tuteur, an internist and pulmonologist, submitted two separate medical evidence reviews, but has never examined Claimant. (DX 25; EX 20-21). In his September 28, 2001 report, he concluded that there was absolutely no convincing information to indicate the presence of CWP or any other coal dust-induced disease, but that Claimant suffered from "industrial bronchitis," which is not typically associated with exposure to coal dust. In his April 22, 2003 review he concluded that based on the totality of available data, there was no convincing information whatsoever to indicate the presence of coal mine dust-induced pulmonary problems. Instead, he opined that Claimant's COPD was the result of childhood pneumonia, evidence of hay fever, and chronic cigarette smoking as an adult. Also, in both reports Dr. Tuteur attempted to undermine the objective findings of other physicians by explaining that the data was more symptomatic of cigarette smoke-induced COPD. For instance, in his 2003 report he stated that if Claimant was suffering from pneumoconiosis, he would expect to find a restrictive abnormality characterized by a reduced total lung capacity instead of the obstructive abnormality with increased total lung capacity as was reflected in Claimant's results. Finally, during his 2004 deposition, Dr. Tuteur stated that there is a possibility that coal mine dust exposure contributed to Claimant's air flow obstruction, but considering the other risk factors, with reasonable medical certainty, he opined that exposure did not contribute in this case.

I do not find Dr. Tuteur's conclusions to be well-reasoned and well-documented. The list of risk factors he relied upon to determine the absence of pneumoconiosis in this case include childhood pneumonia, evidence of hay fever, chronic cigarette smoking, and aspirational

pneumonia. Concerning childhood pneumonia, the only mention in this extensive record is a general reference in Dr. Knight's 10/27/94 report. In that report, however, Dr. Knight found Claimant to be totally disabled due to a combination of previous dust exposure and tobacco usage. (DX 32: 152-155). Also, Dr. Knight's review of the remainder of Claimant's history is very specific, showing the dates of onset. The pneumonia listing states only that it was "childhood." Reoccurrence, extent, severity, or any other reliable documentation that Claimant ever actually had pneumonia as a child is not present. Furthermore, neither of the other examining physicians nor any of the treatment records note any childhood pneumonia. In his deposition, Dr. Tuteur admitted that there is "[l]ittle" evidence in the record concerning childhood pneumonia, and that the data does not say anything about the extent of severity of this occurrence. (EX 26:43-45). Concerning hay fever, Dr. Cohen is the only physician to list the onset of hay fever in 1990, but he does not consider it a factor in his analysis, and concludes that Claimant is totally disabled due to pneumoconiosis and cigarette smoking. (DX 10). Concerning aspiration pneumonia, there was only one incident in January 1991, which occurred in the hospital, was treated immediately, and resulted in clear x-rays within a couple of days. (EX 26). Also, none of Claimant's subsequent records mention any further complications resulting from this episode. Finally, Dr. Tuteur's assessment of the etiology of Claimant's condition does not even discuss the fact that claimant has not smoked for over 20 years and his condition has worsened drastically over that period. In the end, I find that the evidence he used as the basis for his opinion, childhood pneumonia, hay fever, and aspiration pneumonia are tentative at best. Also, I find his statements that there was "absolutely no convincing information" and "no convincing information whatsoever" to link Claimant's condition to coal-dust exposure, is not well-reasoned without an alternative explanation for the deterioration of Claimant's health since he quit smoking 20 years prior.

Dr. Fino, an internist, pulmonologist, and B-reader, submitted a medical evidence review, but has never examined Claimant. (EX 7-8). He found that Claimant suffered from smoking induced COPD and asthmatic bronchitis, but found no objective data of either clinical or legal pneumoconiosis. Also, while Dr. Fino testified that Claimant's lung function and blood gases demonstrate a worsening of his condition since the mid 90's, he forwarded a number of medical studies to support his conclusions. Noting his use of supporting medical literature, I conclude that Dr. Fino's conclusions are well-documented and well reasoned. Therefore, bolstered by his advanced medical credentials, I accord Dr. Fino's opinion probative weight in the determination of whether Claimant suffers from pneumoconiosis.

Dr. Renn, an internist, pulmonologist, and B-reader, submitted a medical evidence review, but has never examined Claimant. (RX 9). He diagnosed Claimant as suffering from chronic bronchitis with an asthmatic component, pulmonary emphysema due to tobacco smoking, and severe to very severe, but significantly bronchoreversible obstructive ventilatory defect. Dr. Renn ruled out coal dust exposure as a cause since (1) Claimant had no radiographic evidence of CWP; (2) Claimant had a greater severity of reduction in his ventilatory function than has been found in any person with CWP; (3) Claimant has hypercarbia which cannot occur in CWP; and (4) Claimant's physiological pattern is consistent with tobacco smoking-induced disease with an asthmatic component, and not CWP. Dr. Renn forwarded a number of medical studies to support his conclusions. Noting his use of supporting medical literature, I conclude that Dr. Renn's conclusions are well-documented and well reasoned. Therefore, bolstered by his

advanced medical credentials, I accord Dr. Renn's opinion probative weight in the determination of whether Claimant suffers from pneumoconiosis.

Dr. Parker, an internist, pulmonologist, and B-reader, submitted a medical evidence review, but has never examined Claimant. (CX 3). Based on PFTs (severe obstruction with minimal acute bronchodilatory responsiveness), ABG studies (non-qualifying but reflecting a pattern of progressive impairment of gas exchange), chest x-rays (negative for opacities), medical treatment records (long-standing, severe COPD and emphysema with symptoms that have become increasingly frequent and severe in recent years), and clinical examinations (rales, rhonchi, crackles, wheezes, and diminished breath sounds were identified at various times which are consistent with chronic lung disease), he concluded that Claimant suffers from pneumoconiosis, manifested as a severe obstructive lung disease, including FEV1 and FEV/FVC ratios, reduced diffusing capacity, and progressively worsening hypoxemia, with normal or hyperinflated lung volumes. While Dr. Parker acknowledged that prolonged tobacco abuse can cause COPD, he also noted that 12 years of underground exposure is also sufficient to cause the disease. And considering the fact that Claimant quit smoking 5-7 years prior to the end of his coal mine work, Dr. Parker attributed Claimant's condition to both coal dust exposure and tobacco smoking. Dr. Parker has set forth clinical observations and findings, and his reasoning is supported by adequate data. Noting his use of supporting medical literature, I conclude that Dr. Parker's opinion is well-reasoned and well-documented. Therefore, enhanced by his credentials as an internist and pulmonologist, I accord Dr. Parker opinion probative weight

The record contains reports by seven doctors, three of which find the existence of pneumoconiosis, and four of which do not. While this claim presents a close call with strong support on both sides, I find that the evidence supports the following conclusions. First, I have found all of the physician's reports, with exception of Dr. Tuteur's to be well-reasoned, documented, and to be accorded probative weight. Second, all of the physicians, with exception of Dr. Mumma, are Board certified internists and pulmonologists. Third, unlike Dr. Knight's earlier opinions, I find that all newly submitted narrative reports do give a clear cut diagnosis of whether Claimant suffers from pneumoconiosis or not. Fourth, Drs. Parker, Cohen and Mumma, relying on objective results, have shown, by a preponderance of the evidence, that Claimant's legal pneumoconiosis has materially worsened since the denial of his 1991 and 1994 claims. This conclusion is further supported by Dr. Fino's admission that he believes that Claimant's lung function and blood gasses have worsened since the mid 90's. (EX 27: 27). Fifth, because of their status as examining physicians, I accord Dr. Altmeyer's and Dr. Cohen's opinions more probative weight than I do the opinions of Drs. Tuteur, Fino, Renn, and Parker. Sixth, since Dr. Cohen's most recent report was issued in 2004, and Dr. Altmeyer has not updated his conclusions since 2001, I find that Dr. Cohen has a more comprehensive grasp on the totality of Claimant's condition at the time of the hearing. Seventh, due to his status as Claimant's last treating physician, I accord Dr. Mumma's opinion controlling weight, especially since his notes, in addition to his report, are replete with consistent, uncontested, notations concerning his pulmonary observations and comparisons that support a worsening condition caused by pneumoconiosis as well as other conditions, and it is supported by the opinions of Drs. Parker and Cohen, who are both internists and pulmonologists. *See Peabody Coal Co. v. Groves*, 277 F.3d 829, 235 n7 (6th Cir. 2002) (the court gave a treating physician's opinion, when buttressed by opinions of other physicians and hospital records, more weight than that given to consulting

or non-examining physicians, on the ground that he has first hand knowledge of the miner's condition). Eighth, I find the opinions of Drs. Cohen, Mumma, and Parker, concerning Claimant's condition more convincing than I do the contrary opinions of Drs. Tutor, Dino and Renn.

Specifically, I find Dr. Parker and Cohen's conclusions concerning the absence of any evidence of asthma to be extremely convincing. They base their opinions, in part, on the fact that while Claimant's COPD progressed in severity over time, and while there were signs of bronchoreversibility, there was only one occasion where this reversibility showed to be significant; and even in that instance, it never reversed to a level greater than severe. Thus, Drs. Cohen and Parker's opinions are supported by the objective test results, and I find them to be highly convincing.

On the other hand, I find it less convincing that Claimant's smoking history, which ended more than 20 years ago, and five to seven years prior to Claimant's retirement from the coal mines, is the sole cause of his continually worsening condition, as Drs. Renn, Tuteur, Fino, and Altmeyer would have me believe, and that coal dust exposure had absolutely no effect on Claimant's condition. Finally, even if Claimant does suffer from asthma, or an asthmatic component compounding his COPD, asthma, asthmatic bronchitis, or emphysema may fall under the regulatory definition of pneumoconiosis if they are related to coal dust exposure. *Robinson v. Director, OWCP*, 3 B.L.R. 1-798.7 (1981); *Trarcik v. Consolidation Coal Co.*, 6 B.L.R. 1-666 (1983). In my judgment, after weighing all of the expert evidence, I find that the Claimant has proven by a preponderance of the evidence that his condition is related to coal dust exposure, and thus has legal pneumoconiosis. Therefore, I find that the sum of the newly submitted medical narrative evidence supports the presence of pneumoconiosis.

The prior claims were denied, in part, because the Director determined that Dr. Knight failed to give a clear cut diagnosis of pneumoconiosis. Dr. Knight's March 26, 1991 opinion stated that Claimant suffered from chronic bronchitis due primarily to cigarette smoking, but he never specifically identified pneumoconiosis. Likewise, Dr. Knight's October 13, 1994 opinion is that Claimant suffers from COPD and chronic bronchitis due to coal mine employment and cigarette smoking. The only abnormal results of Dr. Knight's exam were a PFT showing moderate obstructive ventilatory defect, and distant breath sounds per the physical exam. While I find Dr. Knight's report well-reasoned and documented, due to the remoteness of his examinations and his lack of internist and pulmonologist credentials, I accord his opinion less probative weight than I do the opinions of the other seven more recent physicians.

Therefore, upon comparing the sum of the newly submitted medical narrative evidence against the sum of the previously submitted medical narrative evidence, I find that the newly submitted evidence is more supportive of the claimant, and establishes the presence of pneumoconiosis. Also, I find that the newly submitted medical narrative evidence, including the extensive treatment records, demonstrates that Claimant's condition has worsened as to the presence of pneumoconiosis, and thus, Claimant has succeeded in showing a material change in condition under subsection (a)(4).

I have determined that Claimant has not established the presence of pneumoconiosis under subsection (a)(1), but has shown a material change in condition by proving its existence by a preponderance of the evidence under (a)(4). Therefore, after considering, *de novo*, all of the evidence, both old and new, I find, relying on Drs. Mumma, Cohen, and Parker's evaluations, that the evidence of legal pneumoconiosis established under subsection (a)(4) outweighs the negative x-ray evidence included in subsection (a)(1). Therefore, I find that Claimant has established the existence of pneumoconiosis by a preponderance of the newly submitted and previous medical evidence.

Since Claimant has established a material change in condition in the element of pneumoconiosis, Claimant's subsequent claim will not be denied on the basis of the prior denial. As a result, Claimant must now satisfy the remaining requirements of §718, considering both the old and new evidence, in order to receive benefits.

Arising out of Coal Mine Employment

In order to be eligible for benefits under the Act, Claimant must also prove that pneumoconiosis arose, at least in part, out of his coal mine employment. § 718.203(a). For a miner who suffers from pneumoconiosis and was employed for ten or more years in one or more coal mines, it is presumed that his pneumoconiosis arose out of his coal mine employment. *Id.* As I have found that Claimant has established 12 years of coal mine employment, and as no rebuttal evidence was presented, I find that Claimant's pneumoconiosis arose out of his coal mine employment in accordance with the rebuttable presumption set forth in § 718.203(b).

Total Disability Due to Pneumoconiosis

To prevail, Claimant must also demonstrate that he is totally disabled from performing his usual coal mine work or comparable work due to pneumoconiosis under one of the five standards of § 718.204(b) or the irrebuttable presumption referred to in § 718.204(b). The Board has held that under Section 718.204(b), all relevant probative evidence, both "like" and "unlike" must be weighed together, regardless of the category or type, in the determination of whether the Claimant is totally disabled. *Shedlock v. Bethlehem Mines Corp.*, 9 B.L.R. 1-195 (1986); *Rafferty v. Jones & Laughlin Steel Corp.*, 9 B.L.R. 1-231 (1987). Claimant must establish this element of entitlement by a preponderance of the evidence. *Gee v. W.G. Moore & Sons*, 9 B.L.R. 1-4 (1986).

The amended regulations at § 718.204(c) contain the standard for determining whether Miner's total disability was caused by Miner's pneumoconiosis. Section 718.204(c)(1) determines that a miner is totally disabled due to pneumoconiosis if pneumoconiosis, as defined in § 718.201, is a "substantially contributing cause" of the miner's totally disabling respiratory or pulmonary impairment. Pneumoconiosis is a "substantially contributing cause" of the miner's disability if it has a material adverse effect on the miner's respiratory or pulmonary condition or if it materially worsens a totally disabling respiratory or pulmonary impairment which is caused by a disease or exposure unrelated to coal mine employment. §§ 718.204(c)(1)(i) and (ii). Section 718.204(c)(2) states that, except as provided in § 718.305 and § 718.204(b)(2)(iii), proof that the Miner suffered from a totally disabling respiratory or pulmonary impairment as defined

by §§ 718.204(b)(2)(i), (ii), (iv), and (d) shall not, by itself, be sufficient to establish that the miner's impairment was due to pneumoconiosis.

Except as provided by § 718.204(d), the cause or causes of a miner's total disability shall be established by means of a physician's documented and reasoned medical report. § 718.204(c)(2). The Sixth Circuit Court of Appeals has stated that pneumoconiosis must be more than a "de minimus or infinitesimal contribution" to the miner's total disability. *Peabody Coal Co. v. Smith*, 12 F. 3d 504, 506-507 (6th Cir. 1997). The Sixth Circuit has also held that a claimant must affirmatively establish only that his totally disabling respiratory impairment (as found under § 718.204) was due – at least in part – to his pneumoconiosis. *Cf.* 20 C.F.R. 718.203(a). *Adams v. Director, OWCP*, 886 F.2d 818, 825 (6th Cir. 1988); *Cross Mountain Coal Co. v. Ward*, 93 F.3d 211, 218 (6th Cir. 1996)(opinion that miner's "impairment is due to his combined dust exposure, coal workers' pneumoconiosis as well as his cigarette smoking history" is sufficient). More recently, in interpreting the amended provision at § 718.204(c), the Sixth Circuit determined that entitlement is not precluded by "the mere fact that a non-coal dust related respiratory disease would have left the miner totally disabled even without exposure to coal dust." *Tennessee Consolidated Coal Co. v. Director, OWCP [Kirk]*, 264 F.3d 602 (6th Cir. 2001). A miner "may nonetheless possess a compensable injury if his pneumoconiosis materially worsens this condition." *Id.*

Claimant has not established that he suffers from complicated pneumoconiosis. Therefore, the irrebuttable presumption of § 718.304 does not apply.

Total disability can be shown under § 718.204(b)(2)(i) if the results of pulmonary function studies are equal to or below the values listed in the regulatory tables found at Appendix B to Part 718. All five of the newly and prior submitted pulmonary function tests produced qualifying pre-bronchodilator results. Also, the new evidence also produced qualifying post-bronchodilator results. Bronchodilators were not administered during the 1991 and 1994 tests. Therefore, I find that Claimant has established total disability under subsection (b)(2)(i).

Total disability can be demonstrated under § 718.204(b)(2)(ii) by the results of arterial blood gas studies. There were four arterial blood gas studies performed by examining physicians that were conducted for purposes of litigation. Neither the two newly submitted nor the two prior ABG studies produce qualifying results. Also, there were 45 ABG studies conducted in conjunction with Claimant's medical treatment; 14 qualifying, 31 non-qualifying, and one illegible.

In *Jefferies v. Director, OWCP*, 6 B.L.R. 1-1-13 (1984), the Board held that while ABG studies taken during hospitalization may have been affected by the condition that lead to the miner's hospitalization, and thus unreliable, "without qualified medical testimony to that effect, neither the Board nor the administrative law judge has the requisite medical expertise to make that judgment." *But see Hess v. Director, OWCP*, 21 B.L.R. 1-141 (1998)(it was proper for the administrative law judge to question the reliability of a blood gas study where a physician stated that it was taken while Claimant was in the hospital and "may not be representative of [claimant's] true lung function").

Overall, there are 48 readable ABG studies, 35 of which were negative. Also, all three of the ABG studies since 2001 have been non-qualifying. Furthermore, the three qualifying ABG studies on February 26, 2001 were followed by a non-qualifying ABG on the same date which alludes to a temporary condition that subsequently resolved. Finally, Dr. Cohen, in his January 2, 2004 medical evidence review, stated that the ABG studies performed in conjunction with the treating records were done when Claimant was suffering from exacerbations and are not as useful for evaluating permanent impairment as the studies done for black lung evaluation. Even if the treatment record ABG studies are considered, however, the preponderance of the ABG evidence remains non-qualifying. But, if as Dr. Cohen suggests, the treatment ABG studies are not useful for evaluating permanent impairment, then all of the ABG evidence is non-qualifying. Therefore, I find that Claimant has not established total disability under subsection (b)(2)(ii).

Total disability may also be shown under § 718.204(b)(2)(iii) if the medical evidence indicates that Claimant suffers from cor pulmonale with right-sided congestive heart failure to consider. The record does not contain any evidence of cor pulmonale with right sided-congestive heart failure.¹⁷ Therefore, I find that Claimant has not established the existence of total disability under (b)(3).

Section 718.204(b)(2)(iv) provides for a finding of total disability if a physician, exercising reasoned medical judgment based on medically acceptable clinical or laboratory diagnostic techniques, concludes that Claimant's respiratory or pulmonary condition prevented Claimant from engaging in his usual coal mine employment or comparable gainful employment. Claimant's usual coal mine employment as a general inside laborer required him to lift 35-50 pounds. In addition, his usual employment as a trackman required him to assist another man in loading 25-30, 200-pound ties per day, and to drive in 20 to 26 spikes with a sledgehammer.

The exertional requirements of the claimant's usual coal mine employment must be compared with a physician's assessment of the claimant's respiratory impairment. *Cornett v. Benham Coal, Inc.*, 227 F.3d 569 (6th Cir. 2000). Once it is demonstrated that the miner is unable to perform his usual coal mine work, a *prima facie* finding of total disability is made and the party opposing entitlement bears the burden of going forth with evidence to demonstrate that the miner is able to perform "comparable and gainful work" pursuant to § 718.204(b)(1). *Taylor v. Evans & Gambrel Co.*, 12 B.L.R. 1-83 (1988). Non-respiratory and non-pulmonary impairments have no bearing on establishing total disability due to pneumoconiosis. § 718.204(a); *Jewell Smokeless Coal Corp. v. Street*, 42 F.3d 241 (1994). All evidence relevant to the question of total disability due to pneumoconiosis is to be weighed, with the claimant bearing the burden of establishing by a preponderance of the evidence the existence of this element. *Mazgaj v. Valley Camp Coal Co.*, 9 B.L.R. 1-201 (1986).

Of the eight examining and records review physicians of record, all but Dr. Tuteur concluded that Claimant's chronic respiratory condition was by itself disabling. Dr. Tuteur, however, emphasized the combination of respiratory and other impairments as leading to Claimant's total disability. Also, Dr. Mumma did not make a determination concerning total

¹⁷ I note that there was one reference to "chronic cor pulmonale" in Dr. Mumma's notes dated April 17, 2003. See Appendix A. But, Dr. Mumma's does not provide any follow-up to his "impression," nor is it addressed in any of the other medical reports.

disability. As stated above, I accord more probative weight to the well-reasoned and well-documented opinions of Dr. Cohen and Dr. Altmeyer, who actually examined Claimant, than I do the opinions of the equally credentialed doctors who based their opinions on a records review. As a result, I find that Claimant has proved by a preponderance of the newly and previously submitted medical evidence that he is totally disabled from performing his previous work as a general laborer and trackman, from a respiratory standpoint.

Concerning whether Claimant's disability is due to pneumoconiosis, as I determined above, Drs. Cohen, Altmeyer, Fino, Renn, and Parker's opinions are well-reasoned and well-documented. While Dr. Cohen and Dr. Parker concluded that Claimant's total disability was the result of both his history of cigarette smoking and coal dust exposure, Dr. Altmeyer, Fino, Renn, and Tuteur attributed Claimant's total disability to smoking alone. As explained above, I find the opinion of Drs. Cohen and Parker, concerning the etiology of Claimant's condition more convincing than I do the contrary opinions of Drs. Tutor, Fino, Altmeyer, and Renn. Specifically, I find that the conclusions by Dr. Parker and Dr. Cohen concerning the absence of any evidence of asthma to be extremely convincing. This finding severely undermines the opinions of Drs. Tuteur, Fino, Altmeyer, and Renn. As a result, I find that the sum of the newly and previously submitted medical evidence supports a finding that Claimant's total disability was caused, at least in part, by pneumoconiosis.

Entitlement

As Claimant, Mr. Balsley has established a material change in conditions sufficient to meet the statutory requirements of § 725.309(d), and considering all evidence, old and new, he has established that he has pneumoconiosis arising out of coal mine employment, and is totally disabled due to pneumoconiosis. I find that he is entitled to benefits under the Act. I cannot determine the month of onset of Mr. Balsley's total disability due to pneumoconiosis arising out of coal mine employment. Thus, benefits are payable to Mr. Balsley beginning with the month in which he filed his application for benefits. *See* § 725.503(b). Mr. Balsley filed his application for benefits in November 2000. Therefore, I find that benefits are payable to Mr. Balsley beginning in November 2000.

Attorney's Fees

No award of attorney's fees for services to Mr. Balsley is made herein, since no application has been received from counsel. A period of 30 days is hereby allowed for Mr. Balsley's counsel to submit an application, with a service sheet showing that service has been made upon all parties, including Claimant. The parties have 10 days following receipt of any such application within which to file their objections. The Act prohibits the charging of any fee in the absence of such approval. *See* §§ 725.365 and 725.366.

ORDER

IT IS ORDERED that the claim of Ernest E. Balsley for benefits under the Act is hereby GRANTED.

A

THOMAS F. PHALEN, JR.
Administrative Law Judge

NOTICE OF APPEAL RIGHTS

Pursuant to 20 C.F.R. § 725.481, any party dissatisfied with this Decision and Order may appeal it to the Benefits Review Board within 30 days from the date of this decision, by filing notice of appeal with the Benefits Review Board, P.O. Box 37601, Washington, D.C. 20013-7601. **A copy of a notice of appeal must also be served on Donald S. Shire, Esquire, Associate Solicitor for Black Lung Benefits, Frances Perkins Building, Room N-2117, 200 Constitution Avenue, NW, Washington, D.C. 20210.**

Appendix A

12/18/83 – Examination by Dr. Saunder – Decreased breath sounds with occasional ronchi. He diagnosed Chronic asthmatic bronchitis among other things. (EX 17).

12/19/83 – Examination by Dr. Hillberg – Admitted for back pain. On auscultation of the lungs, breath sounds were somewhat decreased without rales, rhonchi or wheezes or rubs. He diagnosed asthmatic bronchitis, hypertension and chronic anxiety. (EX 17).

2/21/85 – Examination by Dr. Elston – Lungs are clear with decreased inspiratory breath sounds with some prolonged expiratory wheezing. Impression – Probable hypertension. (EX 12).

2/21/85 – [NEW PATIENT REPORT BY DR. ELSTON] – Lungs clear with decreased inspiratory breath sounds with some prolonged expiratory wheezing. Diagnosis: probable COPD. (EX 13).

2/25/85 – X-ray reading by Dr. Greenspan – Lungs are symmetrically well expanded and clear. There are calcified nodes about the left helum. There has been no significant change when compared to the previous supine chest of 9/28/82. Impression: stable chest without signs of acute cardiopulmonary disease. (EX 13).

4/4/85 – Examination by Dr. Elston – Patient had only one asthma attack since last visit. (EX 13).

10/14/85 – X-ray reading by Dr. Mellon – There is thickening of the pleural at the left anterior lung base unchanged from the previous portable study of 2/25/85. The lung fields are clear. (EX 13).

10/17/85 – Examination by Dr. Merz – Admitted for low back strain. Clinical examination of lungs found diminished breath sounds, scattered rhonchi and course rales. Chest x-ray was normal. Diagnosis, Acute lower back strain, hypertension, asthmatic bronchitis. (EX 17).

10/21/85 – Examination by Dr. Saunders – Lungs: decreased breath sounds. Diagnosis: severe lower back strain, asthmatic bronchitis with probable low-grade infection. (EX 17).

11/1/85 – Examination by Dr. Elston – Minimal rhonchi over the area of his rib tenderness. (EX 13).

7/28/86 – Examination by Dr. Elston – Lungs: clear. (EX 13).

11/17/86 – Examination by Dr. Elston – Lungs: decreased breath sounds, scattered rhonchi, no wheeze. Has not smoked for 7 years. (EX 13).

12/30/86 – Examination by Dr. Elston – Lungs: decreased breath sounds, scattered pops and crackle. (EX 13).

1/27/87 – Examination by Dr. Elston – Lungs: decreased breath sounds, no wheezing or rales. (EX 13).

6/1/87 – Examination by Dr. Elston – Lungs: minimally decreased breath sounds. (EX 13).

9/29/87 – Examination by Dr. Elston – Lungs: clear. (EX 13).

10/13/87 – Examination by Dr. Elston – Lungs: clear except for decreased breath sounds. (EX 13).

3/22/88 – Examination by Dr. Elston – Lungs: very decreased breath sounds at the bases. (EX 13).

10/30/89 – Examination by Dr. Elston – Lungs: showed minimally decreased breath sounds. (EX 13).

1/1/90 – Portable chest x-ray revealed lungs to be clear of any infiltrate, edema or pleural fluid. (EX 17).

8/4/90 – CT scan by Dr. Somple – Abnormal CT scan of the brain due to presence of right cerebral hemisphere intracerebral hemorrhage. (EX 17).

8/4/90 – Examination by Dr. Short – Diagnosis: right cerebral hemorrhage with left hemiplegia, but noted that his clinical showed lungs to have very diminished breath sounds bilaterally, but otherwise clear. (EX 17).

8/4/90 – X-ray [portable] reading by Dr. Greenspan – Lungs are clear. No pleural abnormalities are seen. The mediastinum is within normal limits. (EX 17).

8/14/90 – Examination by Dr. Somple – Lungs: scattered left basilar rales. (EX 17).

10/15/90 – ABG by technician Lawton – PCO₂ 44.2 / PO₂ 66.6. (EX 17).

10/15/90 – X-ray [portable] reading – Lungs: clear. No pleural, abnormalities are seen. (EX 17).

11/1/90 – Admitted for issues related to stroke. (EX 17).

11/1/90 – ABG by technician Sheppard – PCO₂ 49.78 / PO₂ 93.8. (EX 17).

11/1/90 – ABG by technician Balsley – PCO₂ 44.2 / PO₂ 67.8. (EX 17).

11/1/90 – ABG by technician Balsley – PCO₂ 42.8 / PO₂ 44.8 – PO₂. Within critical limits. (EX 17).

12/21/90 – Examination by Dr. Elston – No chest pain. Shortness of breath at times but not unusual. Still has a lot of phlegm. Lungs: clear. (EX 13).

1/24/91 - X-ray reading by Dr. Boyse – No signs of pulmonary or pleural disease. (EX 13).

1/25/91 – X-ray [portable] reading by Dr. Boyse – Vague density behind the heart as compared to previous film the same day. (EX 17).

1/25/91 – X-ray [portable] reading by Dr. Boyse – Lungs: clear. (EX 17).

1/25/91 – ABG by technician Sheppard – PCO2 36.3 / PO2 127.5. (EX 17).

1/25/91 – ABG by technician Sheppard – PCO2 28.1 / PO2 168.1. (EX 17).

1/25/91 – ABG by technician Blake – PCO2 37.1 / PO2 49.1 – PO2. Within critical limits. (EX 17).

1/25/91 – ABG by technician Sheppard – PCO2 28.7 / PO2 92. (EX 17).

1/26/91 – X-ray [portable] reading showed no change from previous day. (EX 17).

1/26/91 – ABG by technician Balsley – PCO2 35.3 / PO2 103.7. (EX 17).

1/26/91 – ABG by Technician Moorehead – PCO2 38.1 / PO2 84.2. (EX 17).

1/26/91 – ABG by technician Moorhead – PCO2 38.7/PO2 80.4. (EX 17).

1/26/91 – ABG by technician Decker – PCO2 39.1 / PO2 79.9. (EX 17).

1/26/91 – ABG by technician Decker – PCO2 39.4 / PO2 73.4. (EX 17).

1/27/91 – ABG by technician Lawton – PCO2 41.4 / PO2 79.9. (EX 17).

1/27/91 – ABG by technician Lawton – PCO2 41.9 / PO2 89.4. (EX 17).

1/27/91 – ABG by technician Balsley – PCO2 42.7 / PO2 134.1. (EX 17).

1/27/91 – X-ray reading by Dr. Boyse – The follow up film on the 27th at this time reveals improvement at the left base. There is no longer a double pleural line behind the heart indicating that there has been a definite change or improvement. There appears to be a resolved left lower lobe atelectasis since the last examination. Impression : Normal (EX 13).

1/28/91 – X-ray [portable] reading by Dr. Steinberger – There is an acute infiltrate in the right lower lobe. This is a new finding. The left lung is clear. The upper mediastinum appears widened to the right. This could be rotational and in part due to the portable technique. No other abnormalities are identified. Impression: acute infiltrate right lower lobe. (EX 17).

1/28/91 – ABG by technician Decker – PCO2 40.4 / PO2 88.6. (EX 17).

1/28/91 – ABG by illegible technician – PCO₂ 38.2 / PO₂ 53.4. PO₂ is within critical limits. (EX 17).

1/31/91 – X-ray reading – Comparison to 1/28/91 x-ray study shows the previously described basilar infiltrate has cleared. There appears to be some blunting of the left costophrenic angle, unchanged. Conclusion: clearing of previously described right basilar infiltrate. (EX 17).

1/31/91 – ABG by technician Moorehead – PCO₂ 42.7 / PO₂ 54.6. PO₂ is within critical limits. (EX 17).

1/31/91 – Radiology consultation by Dr. Graber – The previously described basilar infiltrate has cleared. There appears to be some blunting of the left costophrenic angle, unchanged. (EX 13).

2/4/91 – ABG by technician Moorehead – PCO₂ 35.5 / PO₂ 61.7. (EX 17).

2/22/91 – Examination by Dr. Elston – Lungs: showed scattered rhonchi on the right. (EX 13).

3/8/91 – X-ray reading by Dr. Greenspan – The lungs are emphysematous in configuration. There are signs of bronchial wall thickening but without discrete infiltrate. Impression: Emphysematous chest with bronchial wall thickening but without discrete infiltrate. (EX 13).

7/16/91 – ABG by illegible technician – PCO₂ 41.2 / PO₂ 77.9. (EX 13).

7/30/91 – Examination by Dr. Elston – Lungs: clear. Looks quite good. (EX 13).

9/11/91 – X-ray reading by Dr. Steinberger – There is a hazy density in the left base near the diaphragm. This may be a pleural reflection in as much as it is visible on a previous film of 6/26/91. The lungs are free from infiltrate otherwise. No effusion or other sign of active disease. (EX 13).

9/16/91 – Discharge summary by Dr. Elston after 9/11 admission due to cough productive of purulent sputum and increased shortness of breath – Chest x-ray showed no active infiltrate. Diagnosis: bronchitis and COPD with acute exacerbation. (EX 17).

10/18/91 – Examination by Dr. Elston – Lungs: clear, no chest pain, and no edema. (EX 13).

2/29/92 – Emergency department report by Dr. Ramey's – URI symptoms, shortness of breath, and a non-productive cough. Chest examination revealed diffuse and expiratory wheezes, chest x-ray revealed some interstitial fibrosis. Conclusion: patient suffered from COPD with exacerbation. (EX 17).

3/1/92 – ABG – Illegible. (EX 17).

4/2/92 – X-ray reading by Dr. Elston – Lungs appear mildly emphysematous but otherwise are free of infiltrate. There is some pleural thickening at the left base. No significant change since 2/29/92. Impression – Stable emphysematous chest without signs of acute cardiopulmonary disease. (EX 17).

4/9/92 – ABG by a technician Moorehead – PCO2 38.6 / PO2 79.2. (EX 17).

9/15/93 – X-ray reading by Dr. Boyse – Lungs were expanded and clear. No evidence of active disease. (EX 17).

10/6/93 – ABG by a technician Raueh – PCO2 38.3 / PO2 74.4. (EX 17).

10/11/93 – X-ray [portable] reading by Dr. Graber – No significant infiltrates. (EX 17).

10/21/93 – X-ray [portable] reading by Dr. Greenspan – Lungs are well expanded and free of infiltrate, and he did not see a significant change when compared to the 10/11/93 examination. (EX 17).

11/4/93 – ABG by an illegible technician – PCO2 41 / PO2 71.9. (EX 17).

11/4/93 – X-ray reading – No active cardiopulmonary disease and no change since the previous x-ray. (EX 17).

11/4/93 – Emergency room report by Dr. Charnes – Diagnosis: acute bronchitis. (EX 17).

11/4/93 – X-ray reading by Dr. Graber – The lung fields are clear. There is no pleural effusion or overt congestive failure. There is no cardiopulmonary disease and no change since the previous study of 10/21/93. Lung scan by Xenon gas, and reveals that there was a significantly patchy distribution of activity in both lung fields during the inspiratory phase. There is moderate diffuse air trapping in the expiratory phase. The perfusion study was also abnormal showing diffuse, patchy appearance of both lungs in a similar distribution to the ventilation study. Conclusion: Matching ventilation and perfusion abnormalities, compatible with low probability for pulmonary embolus. (EX 13).

2/13/94 – X-ray reading by Dr. Safko – There is no pleural fluid present. The lungs are clear. There is no change from a previous film of 11/4/93. Impression: No active disease shown. (EX 13).

2/13/94 – Examined by Dr. Schuster – Conclusion: COPD with acute exacerbation. Lungs: fairly diffuse expiratory wheezes and a few rales. (EX 17).

2/14/94 – X-ray reading by Dr. Safko – There is no pleural fluid present, the lungs are clear. No change since 11/4/93 film. No active disease shown. (EX 17).

2/14/94 – Examined by Dr. Elston – Conclusion: COPD and asthmatic bronchitis, and clinical examination of the lungs showed 1+ expiratory wheeze, scattered rhonchi. (EX 17).

7/15/94 – Emergency room report by Dr. Shara – Chest is clear with bronchial breath sounds, and a prolonged expiratory phase. X-ray showed negative for infiltrate. Infectious bronchitis was diagnosed. (EX 17).

7/15/94 – Examination – Lung fields are free of active infiltrate. There are a number of calcified nodes in the left hilum. There is no pleural effusion and no gross congestive failure. There is no active cardiopulmonary disease. (EX 17).

8/2/94 – Admitted for shortness of breath. (EX 17).

8/2/94 – ABG by technician Long – PCO₂ 42 / PO₂ 71.5. (EX 17).

8/2/94 – X-ray reading – Lung fields remain clear. (EX 17).

8/4/94 – X-ray reading by Dr. Graber – Lung fields appear fairly well aerated. There is no gross infiltrate, although there may be a very slight degree of increased interstitial density at the right base. (EX 17).

8/5/94 – Examination by Dr. Elston – Patient suffers from COPD with acute exacerbation and possible early right basilar pneumonia. (EX 17).

10/24/94 – X-ray reading by Dr. Graber – Comparison is made with the previous study of 10/13/94. The lungs appear mildly emphysematous. I see no other active infiltrates. Conclusions: Mildly emphysematous-appearing chest with no acute changes. (EX 13).

10/26/94 – Emergency room report by Dr. Writesel – Complaining of dyspnea and anterior chest pain. Lungs produced breath sounds are coarse bilaterally with questionable faint rales in the left base posteriorly. Otherwise, good air movement. Diagnosis: left lower lobe infiltrate. (EX 17).

10/26/94 – X-ray reading by Dr. Graber – Mildly emphysematous-appearing chest with no acute changes. (EX 17).

1/6/95 – Emergency room examination by Dr. Ramey – Shortness of breath and harsh productive cough. Patient was somewhat dyspneic at rest, suffered from intermittent harsh cough, and had diffuse coarse sonorous rhonchi. He diagnosed COPD with acute exacerbation and acute bronchitis. (EX 17).

1/6/95 – ABG by technician Long – PCO₂ 44 / PO₂ 100.8. (EX 17).

1/6/95 – X-ray reading by Dr. Steinberger – No evidence of active disease. (EX 17).

1/6/95 – X-ray reading by Dr. Steinberger – Lungs are expanded and clear. There is no evidence of active disease. (EX 13).

1/9/95 – ABG by technician Stewart – PCO₂ 42 / PO₂ 66.5. (EX 17).

1/27/95 – Examination by Dr. Elston – Lungs show markedly decreased breath sounds. No wheezing or rales. Patient wants to go back to work. (EX 13).

2/13/95 – ABG by technician Long – PCO₂ 42.5/PO₂ 58.9. (EX 17).

2/13/95 – X-ray reading by Dr. Greenspan – Lungs are emphysematous, but free of acute infiltrate when compared to previous study of 1/6/95. There are signs of bronchial wall thickening but this is actually less prominent. Diagnosis: Emphysematous chest without acute infiltrate. (EX 17).

2/15/95 – Examination by Dr. Ramey – Diagnosis: COPD with exacerbation and Bronchitis. (EX 17).

2/15/95 – X-ray reading by Dr. Greenspan – Lungs are emphysematous, but free of acute infiltrate when compared to previous study of 1/6/95. There are signs of bronchial wall thickening but this is actually less prominent. The heart is not enlarged. Impression: Emphysematous chest without acute infiltrate. (EX 13).

4/11/95 – Examination by Dr. Elston – Lungs have diminished breath sounds bilaterally. He has trace to 1+ bilaterally wheezes. I suspect he has a mild COPD exacerbation caused by acute bronchitis. (EX 13).

4/11/95 - X-ray reading by Dr. Graber – No pleural fluid is shown on this study. The lungs appear to be clear with no infiltrates or masses. The heart and mediastinum are unremarkable. There is no change from an old study. The lungs are mildly emphysematous. Impression: mild emphysema, no change from old study. (EX 13).

4/11/95 – X-ray reading – No active intrathoracic disease. (EX 17).

4/11/95 – ABG by illegible technician – PCO₂ 37.6 / PO₂ 61.2. (EX 17).

5/4/95 – Examination by Dr. Elston – Decreased breath sounds. Impression: COPD and other ailments. (EX 13).

5/11/95 – PFT by Dr. Branditz – No values or tracings in file. Impressions: Severe COPD pattern. There is a very severe degree of functional impairment demonstrated. There is significant improvement in expiratory flow rates noted after the administration of an inhaled bronchodilating agent. Function impairment remains very severe even after this improvement. Inspiratory flow rates are normal. Lung volumes are somewhat elevated with a significantly increased residual volume measurement. This would be consistent with pulmonary air trapping from the above noted obstructive lung disease. Airways resistance values are markedly abnormal. The diffusing capacity for carbon monoxide is decreased. Again this would be consistent with the above noted abnormalities. Effort and cooperation were good. (EX 13).

7/10/95 – X-ray reading by Dr. Mellon – The lungs are clear and unchanged from 4/11/95. No active intrathoracic disease or change. (EX 13).

7/9/95 – X-ray reading by Dr. Graber – No definite active infiltrates, no pleural effusions, and there were no acute findings noted. (EX 17).

7/9/95 – X-ray reading by Dr. Mellon – Lungs are clear and unchanged. (EX 17).

7/28/95 – Emergency department report by Dr. Rossi – Diagnosis: acute bronchitis. (EX 17).

9/2/95 – Emergency department report by Dr. Watson – Complaint for possible pneumonia. Some diffuse rhonchi and some expiratory wheezing. Diagnosis: COPD mild acute exacerbation with bronchitis. (EX 17).

9/2/95 – X-ray reading by Dr. Boyse – No pulmonary abnormality and no specific findings. (EX 17).

9/2/95 – ABG by technician Stewart – PCO₂ 33.3/PO₂ 65.3. (EX 17).

9/3/95 – Admitted for shortness of breath and other symptoms. (EX 17).

9/28/95 – Examination by Dr. Elston – Lungs: clear. (EX 13).

9/28/95 – Admission report by Dr. Elston – Admitted at emergency room for illness, symptoms included shortness of breath, and non-productive cough. Diagnosis: probable pneumonia with co-existent COPD. (EX 17).

9/28/95 – ABG by technician McCloud – PCO₂ 30.1/PO₂ 60.7. (EX 17).

9/28/95 – X-ray reading by Dr. Safko – Lungs are clear with no fluid. (EX 17).

9/29/95 – X-ray reading by Dr. Safko – The lungs are clear with no fluid. There is some slight increase in interstitial markings in the lower lung fields. (EX 13).

10/3/95 – ABG by technician Moorehead – PCO₂ 43.9 / PO₂ 65.2. (EX 17).

10/4/95 – Discharge summary report – Diagnosis: pneumonia, COPD with mild exacerbation, and mild hyponatremia. (EX 17).

10/21/95 – X-ray reading by Dr. Watson – No infiltrates. ABG study resulted in values – PO₂ 61.6 / PCO₂ 31.7. (EX 17).

10/21/95 – X-ray reading by Dr. Graber – Compared to a 9/28/95 x-ray, lung fields were clear, no pleural effusions, no active cardiopulmonary disease. (EX 17).

11/6/95 – Admission examination by Dr. Elston – Admitted for shortness of breath, weakness, and fever. Clinical examination showed distressed breath sounds with scattered rhonchi. Chest x-ray was clear. (EX 17).

11/6/95 – Discharge report by Dr. Valentine – Diagnosis: acute exacerbation of COPD. (EX 17).

11/6/95 – ABG by technician Long – PCO₂ 31.6 / PO₂ 61.7. (EX 17).

12/9/95 – Examination by Dr. Elston – Diffuse wheezing with decreased inspiratory breath sounds. (EX 13).

2/6/96 – Examination by Dr. Elston – Decreased breath sounds but clear. (EX 13).

2/22/96 – Examination report by Dr. Ramey – Shortness of breath. Lungs: 1+ expiratory wheeze. Diagnosis: COPD with exacerbation status post CVA with left hemiplegia. (EX 17).

2/22/96 – ABG by technician Stewart – PCO₂ 38.7 / PO₂ 63.5. (EX 17).

2/22/96 – X-ray reading by Graber – Lung fields clear, no pleural effusion or gross congestive failure, no active cardiopulmonary disease. (EX 14).

2/25/96 – Emergency Department report by Dr. Korn – Lungs: clear. (EX 14).

3/3/96 – X-ray reading by Dr. Boyse – AP, upright and lateral views of the chest reveal a normal size heart. The lungs are clear of any infiltrate, edema or pleural fluid. Impression: no pulmonary abnormality. (EX 13).

3/10/96 – Emergency department report by Dr. Keseg – Admitted for abdominal pain with an unknown etiology. Lungs were fairly clear to auscultation bilaterally, and there was no significant decreased breath sounds, rhonchi, or wheeze. (EX 17).

3/10/96 – X-ray reading by Dr. Graber – Lung fields are clear, and no active cardiopulmonary disease. (EX 17).

3/11/96 – X-ray reading by Dr. Graber – Comparison to previous study of 9/2/95. The lung fields are clear and the heart and mediastinal structures are intact. There is no pleural effusion. No gross congestive failure. Conclusion: No active cardiopulmonary disease. (EX 13).

3/12/96 – X-ray reading by Dr. Boyse – Lungs are clear of any infiltrate, edema or pleural fluid. No pulmonary abnormality. (EX 17).

3/12/96 – Readmission report by Dr. Merz – Lungs: breath sounds diminished, a few rhonchi. No rales or dullness. Diagnosis: Poorly controlled diabetes, severe COPD, hypertension. (EX 17).

3/18/96 – Discharge report by Dr. Elston – X-ray negative. Diagnosis: Type II diabetes out of control, severe COPD. (EX 17).

4/29/96 – Examination by Dr. Elston – Lungs have decreased breath sounds, but no wheeze. (EX 13).

6/5/96 – Examination by Dr. Elston – Lungs: clear. (EX 13).

8/12/96 – Examination by Dr. Elston – Lungs have decreased breath sounds with no wheeze. (EX 12).

9/5/96 – Emergency Department report by Dr. Mazza – Admitted for shortness of breath. Diagnosis: exacerbation of COPD, and found the lungs to show significant decreased air movement with some scattered wheezing throughout. (EX 17).

9/5/96 – X-ray reading by Dr. Mellon – Lungs are clear of acute infiltrate. No active intrathoracic disease. (EX 17).

9/11/96 – Emergency Department report by Dr. Ramey – Lungs: reveal decreased breath sounds with scattered wheezes. Impression: COPD with bronchospasm, diabetes, hypertension, status post CVA with left hemiplegia. (EX 17).

9/11/96 – Admission report by Dr. Merz – Admitted and diagnosed with COPD. Lungs: diminished breath sounds with scattered rhonchi and wheezes, no rales or dullness. Diagnosis: COPD, decompensated, probably pneumonia; possibly some element of LV failure in view of his orthopnea and recent increase in his dyspnea, and other things. (EX 14).

9/11/96 – Examination by Dr. Ramey – Decreased breath sounds with scattered wheezes. Diagnosis: COPD with bronchospasm. (EX 14).

9/11/96 – ABG by technician Long – PO₂- 72 PCO₂- 39. (EX 14).

9/12/96 – X-ray reading by Dr. Boyse – Normal. (EX 14).

9/12/96 – X-ray [portable] reading by Dr. Boyse – Normal heart size with no pulmonary infiltrate, edema or pleural fluid. There is no change from a previous examination of 9/6/96. Impression: normal portable examination. (EX 13).

9/24/96 – Examination by Dr. Elston – Lungs show markedly decreased breath sounds. He has no wheezing. Impression: COPD severe, with mild exacerbation. (EX 13).

10/1/96 – Examination by Dr. Elston – Moving air much better, no wheezing and his aeration is much better. (EX 13).

10/3/96 – Discharge examination by Dr. Merz – Diminished breath sounds with scattered ronchi and wheezes. Also, old left hemiplegia was present. Chest x-ray was normal. (EX 14).

11/4/96 – Examination by Dr. Elston – Dyspneic with any exertion. Lungs are clear with good air movement. Somewhat decreased breath sounds. His COPD is quite severe, although he does not yet require home oxygen. (EX 13).

11/8/96 – X-ray reading by Dr. Muchnok – The lung fields are clear and demonstrate no acute change from 9/11/96 exam. Impression: no acute process. (EX 13).

2/25/97 – Examination by Dr. Elston – Lungs show no wheezing and a decreased breath sound. Impression is severe COPD and other ailments. (EX 13).

5/15/97 – X-ray reading by Dr. Greenspan – The lungs are emphysematous in appearance but similar to the previous study of 11/8/96. No acute infiltrate is noted. Impression: Emphysematous chest without acute findings radiographically. (EX 13).

7/3/97 – Examination by Dr. Elston – Lung: fairly clear with decreased breath sounds. (EX 13).

10/21/97 – Examination by Dr. Elston – Lung: markedly decreased breath sounds. Minimal expiratory wheeze, and scattered Rhonchi. Impression: severe COPD, and other ailments. (EX 13).

1/5/98 – Examination by Dr. Elston – Lung: markedly decreased breath sounds but clear. Impression: probable eczema on the chest vs. seborrhea, acute bronchitis. (EX 13).

2/18/98 – Emergency department report by Dr. Crouch – Scattered wheezes, x-ray showed no acute infiltrate or mass. Diagnosis: acute bronchitis and upper respiratory infection. (EX 14).

2/18/98 – X-ray reading by Dr. Mellon – Lungs are clear and unchanged from 11/15/97. No active intrathoracic disease. (EX 14).

2/23/98 – Admitted for Exacerbation COPD. (EX 14).

2/23/98 – X-ray reading by Dr. Graber – No definite active cardiopulmonary disease. (EX 14).

2/23/98 – ABG by technician Moorehead – PCO₂ 36.8 /PO₂ 60.1. (EX 14).

2/23/98 – Examination by Dr. Elston – Lungs: 1-2+ expiratory wheezes with a markedly decreased inspiratory breath sounds and diffuse rhonchi. (EX 14).

2/26/98 – Discharge report by Dr. Elston – X-ray showed no acute cardiopulmonary disease, and ABG showed PCO₂ 37/PO₂ 60 – Diagnose COPD, acute exacerbation, resolving acute bronchitis, and other things. (EX 14).

3/10/98 – Examination by Dr. Elston – Lungs: decreased breath sounds, but clear. Impression: COPD. (EX 13).

3/18/98 – Admission report by Dr. Andras – Breathing problems. Assessment: acute exacerbation of COPD and bronchitis. Chest x-ray showed emphysema but clear lung fields, no change from x-ray 2 weeks earlier. (DX 27).

3/18/98 – X-ray reading by Dr. Charles Muchnok – No acute process. (DX 27).

3/21/98 – Examination by Dr. Short – Lungs have 1+ expiratory wheeze throughout, no rales. Chest x-ray is clear. Impression: acute bronchitis. (EX 19).

8/2/98 – Emergency admittance due to chest pain, cough and yellowish phlegm. (DX 27).

8/2/98 – Emergency department report by Dr. Andras – Chest tightness. Lung fields have markedly diminished breath sounds bilaterally with virtually no audible breath sounds anteriorly and some faint expiratory wheezing posteriorly. EKG shows normal rhythm and x-ray shows hyperinflated dark lung fields with no infiltrates or other abnormalities. No acute change was noted. Diagnosis: exacerbation of COPD and bronchitis. (EX 14).

8/2/98 – X-ray reading by Dr. Muchnok – Since the previous study, some increased density has developed involving the medial right base seen with certainty only on the frontal view. Requested a repeat x-ray to confirm as this is not definitely present on the lateral view. (EX 14).

8/3/98 – X-ray reading by Dr. Muchnok – Comparison is made to a 3/21/98 exam. There is some questionable increased density involving the right medial base seen with certainty only on the frontal view. I am uncertain if this is a real finding or not. The arms are present over a portion of the chest on the lateral view but I cannot see a corresponding infiltrate laterally. Nonetheless, the change in the appearance of the chest, on the frontal view, is somewhat concerning. I suppose this could be artifactual but given the patient's symptoms, I am concerned for infiltrate. (EX 13).

8/5/98 – X-ray reading by Dr. Boyse – There is no density or abnormality seen in the right base. It is possible the previous density is caused by the AP lordiotic projection of the chest which has caused extrinsic soft tissue to appear abnormal over the right base. Impression: Normal at this time. (EX 13).

8/26/98 – Examination by Dr. Elston – Lungs: clear with markedly decreased breath sounds. He has minimal expiratory wheeze only with forced expiration. (EX 13).

10/28/98 – Admitted for shortness of breath and tightening lungs. (EX 13).

10/28/98 – Examination by Dr. Parrett – Lungs have diminished breath sounds, and virtually no air is heard moving throughout the lungs. He diagnosed COPD with near syncope likely related to hypoxemia. (EX 14).

10/29/98 – Admitted for COPD with exacerbation. (EX 14).

10/29/98 – ABG by illegible technician – PCO₂ 39.2 / PO₂ 79.2. (EX 14).

10/30/98 – Discharge summary by Dr. Elston – X-ray showed no acute disease, and diagnosed COPD, acute exacerbation, among other things. (EX 14).

11/3/98 – [NEW PATIENT EXAMINATION BY DR. KALIS] – Breathing sounds are diminished, but fairly clear. (EX 12).

12/4/98 – Examination by Dr. Kalis – There are diffuse rhonchi and occasional wheezes. No rales are heard. Assessment: **COPD secondary to cigarette smoking.** (EX 12).

12/27/98 – X-ray reading by Dr. Graber – Compared to study from 10/28/98 and determined that there was no definite active cardiopulmonary disease. (DX 27).

12/27/98 – X-ray reading by Dr. Graber – Lung fields are clear. No definite active cardiopulmonary disease. (EX 14).

12/27/98 – ABG by technician Pitner – PCO₂ 36 / PO₂ 71.8. (EX 14).

12/28/98 – X-ray [portable] reading by Dr. Graber –Increased opacification of the right macillary sinus which could be due to either fluid or mucosal thickening. (DX 27).

12/28/98 – Examination by Dr. Clemens – Admitted for exacerbation of COPD. Lungs were markedly decreased breath sounds throughout. Occasional rhonchi, no crackles. Chest x-ray showed no acute or active cardiopulmonary disease, no pleural effusion and no infiltrates. (EX 14).

12/28/98 – Examination by Dr. Allen – Chest has inspiratory rhonchi heard throughout with bilateral expiratory wheezing. There was no focal consolidation. ABG was PCO₂ 36/ PO₂ 72. Diagnosis: chest pain and COPD exacerbation. (EX 14).

1/1/99 – Discharge summary by Dr. Elston – Diagnosis: COPD, acute exacerbation, possible acute bronchitis, and maxillary sinusitis. (EX 14).

1/27/99 – Examination by Dr. Kalis – Since last year he was hospitalized for his COPD with what sounds like purulent bronchitis. He is feeling better since that time, but is still coughing up some yellow sputum. He did notice some shortness of breath last night. Lungs are clear at this time and no rhonchi or wheezes are heard. He has severe COPD. (EX 12).

3/25/99 – Examination by Dr. Kalis – Lungs are very clear today with no wheezes, rhonchi or rales noted. COPD is stable. (EX 12).

6/24/99 – Examination by Dr. Kalis – Lungs are in essence clear. COPD is doing nicely. (EX 12).

8/15/99 – Emergency Department report by Dr. Gaffeo – Lungs show decreased breath sounds throughout with some occasional rhonchi. (EX 19).

10/21/99 – Examination by Dr. Kalis – Although he has bronchitis right now with yellow sputum, he is basically doing well. He has not had wheezing or shortness of breath. (EX 12).

2/22/00 – Examination by Dr. Kalis – He has been feeling fairly well and has no real shortness of breath or sputum production. Lung examination shows lungs are virtually clear. COPD is doing well. (EX 12).

6/29/00 – Examination by Dr. Kalis – He is using a combination of Albuterol and Atrovent in an aerosol machine, about three or four times a day. He is doing nicely with this. He uses Servent at nighttime. He has done very well with this regimen. He has not has an exacerbation of this lung disease. He has no wheezing or shortness of breath. Lung examination shows clear. Diagnosis. COPD, currently quiescent. (EX 12).

8/29/00 – X-ray [portable] report by Dr. Elk – No acute infiltrate, pleural effusions or pneumothrax . No acute abnormality detected. (EX 12).

8/29/00 – Emergency Department report by Dr. Feicht – Admitted for chest pain. Lungs: clear but diminished without rales, rhonchi or wheezes. X-ray showed lungs are symmetrically well-expanded and clear. No significant pleural or parenchymal abnormalities. There is no radiological evidence of active disease. (EX 19).

8/29/00 – Examination – Conclusion: lungs appear emphysematous, and there is some questionable increased right medial base markings identified that could represent a developing infiltrate. He diagnosed COPD. (DX 27).

8/30/00 – Stress test by Dr. Brantley – Test based on sharp atypical chest pain of undermined etiology. X-ray views were not interpretable secondary to the poor sound wave transmission, related to patient's COPD. Impression: Normal Dobutamine stress echocardiogram, and low probability for clinically significant coronary artery disease. (EX 19).

8/30/00 – Examination by Dr. Kalis – Chest shows clear to auscultation and percussion. This is probably non cardiac chest pain. Stress test was negative and patient was discharged. (EX 19).

10/17/00 – Examination by Dr. Kalis – No real problems with cough, chest colds or shortness of breath, etc. He does not hear himself wheezing. Chest examination shows COPD. Largely doing nicely on the current medicinal regimen. (EX 12).

2/10/01 – Examination by Dr. Kalis – He recently had a bout of bronchitis. He was given Cipro and is doing better. His sputum has cleared. He still feels short of breath. Lungs have a distant breath sounds, but largely wheeze free. No rhonchi or wheezes. Conclusion, bronchitis, in a patient with severe chronic lung disease, doing fairly well. (EX 12).

2/26/01 – X-ray [portable] reading by Dr. Muchnok – Lungs appear emphysematous. There is some questionable increased right medial base markings identified that could represent a developing infiltrate. Impression: COPD, questionable right medial base infiltrate. (EX 12).

2/26/01 – Emergency Department Report by Dr. Kelly – Admitted for breathing difficulty. Lungs showed poor air movement with scattered moderate inspiratory and expiratory wheezes.

Positive accessory muscles. X-ray shows a faint infiltrate at the right cardiac silhouette. Impression: COPD exacerbation and pneumonia. (EX 19).

2/26/01 – Examination by Dr. Kalis – Diffuse bilateral wheezes and rhonchi, no rales. Assessment: asthmatic bronchitis in a patient with known COPD. (EX 19).

2/26/01 – Examination by Dr. Kelly – Admitted because unremitting asthma that was unsuccessfully treated as an outpatient. Further treatment and evaluation per Dr. Kalis. Impression: COPD exacerbation and pneumonia. (DX 27).

2/26/01 – Examination by Dr. Kalis – Chest x-ray showed a hazy density at the medial right lung bases, probably related to a cardiophrenic fat pad, but there was no change from the previous films. Diffuse bilateral wheezes and rhonchi. ABG showed PCO₂ 32, PO₂ 62. Impression: asthmatic bronchitis in a patient with known severe COPD/diabetes mellitus/hypertension/ old CVA. (DX 27).

2/26/01 – X-ray reading by Dr. Mellon – No change from 2/26/01 x-ray. (DX 27).

2/26/01 – ABG interpretation by Dr. Kelly – PCO₂ 32.3 and PO₂ 62.7. (DX 27).

3/1/01 – X-ray reading by Dr. Mellon – Mild hazy density at the medial right lung base which appears unchanged from the portable of 2/26/01 and AP and lateral chest film of 12/27/98. This, at least in part, is probably related to cardiophrenic fat pad. Lungs are otherwise clear. (EX 12).

3/4/01 – Discharge summary by Dr. Kalis – Asthmatic bronchitis in a patient with known COPD. (EX 19).

3/6/01 – Examination by Dr. Kalis – Patient called because very short of breath. Can't walk from bathroom to living room. Coughed up almost black sputum prior night. (EX 12).

3/12/01 – Examination by Dr. Kalis – Patient recently hospitalized for a bout of severe asthmatic bronchitis without pneumonia. Since discharged, no wheezing, but still coughing up some yellow sputum. Lung sounds are distant, but no wheezes. A few rhonchi. Asthmatic bronchitis is resolving. (EX 12).

6/26/01 – Examination by Dr. Kalis – Concerning COPD, no wheezes or ronchi. He gets an occasional upper respiratory infection and we treat him with antibiotics. Thus far, he has done well. Conclusion – suffers from COPD, but no current problem. (EX 12).

7/8/01 – Emergency Department report by Dr. Kerns – Admitted for shortness of breath. Exam showed lungs to be diminished in all fields but fairly clear, and no obvious wheezes, rales or rhonchi. Portable chest shows hyperinflation, but no obvious CHF or infiltrates. Impression: mild COPD exacerbation. (EX 19).

7/8/01 – Admission report by Dr. Kerns – Admitted due to shortness of breath. Claimant's lungs were diminished in all fields, but fairly clear. No obvious wheezes, rales or ronchi. EKG: normal sinus rhythm. Portable chest x-ray shows hyperinflation. Impression: mild chronic obstructive pulmonary disease exacerbation. (DX 27).

7/8/01 – X-ray reading by Dr. Boyse – No pulmonary abnormality at that time. (DX 27).

10/26/01 – Emergency Department Report by Dr. Kerns – Lungs: expiratory wheezes in all fields. He is not using intercostals muscles for respiratory support. Chest x-ray shows right lower lobe inflation. Diagnosis: pneumonia, COPD exacerbation, mild dehydration. (EX 24).

10/26/01 – X-ray reading by Dr. Mellon – Infiltrate in the right lower lung field which is new since the portable film of 7/8/01. Left lung is clear. (EX 24).

10/27/01 – Examination by Dr. Kalis – Diagnosis: pneumonia in a patient with severe COPD, as well as other ailments. (EX 24).

10/28/01 – Examination by Dr. Kalis – Admitted with pneumonia and mild hypoxemia. X-ray showed infiltrate in the right lower lung field. Diagnosis: pneumonia and COPD. Patient was discharged. (EX 24).

11/7/01 – Examination by Dr. Kalis – Pneumonia resolved, doing nicely. (EX 12).

11/7/01 – X-ray reading by Dr. Kalis – PA and lateral chest x-ray show the bony structures to be normal. The cardiac silhouette is unrevealing. The lung fields show some mild accentuated markings in the right base and left base. No old films are available for comparison at this time. This may represent chronic fibrosis. (EX 12).

12/18/01 – Examination by Dr. Henzes – Admitted due to shortness of breath. X-ray showed no evidence of infiltrate or effusion. Diagnosis: COPD exacerbation. (EX 24).

12/18/01 – Examination by Dr. Kalis – Diagnosis: asthmatic bronchitis in a patient with underlying COPD. (EX 24).

12/18/01 – X-ray [portable] reading Richard Folke – Some hyperinflation of the lungs. The left lung is grossly clear. There is mild nonconfluent interstitial prominence in the right lower lung. There is no confluent pulmonary consolidation. Conclusion: Mild interstitial prominence/minimal infiltrate in the right lower lung. (EX 24).

12/20/01 – Discharge summary by Dr. Kalis – Chest x-ray showed mild interstitial prominence/minimal infiltrate in the right lower lung. Patient admitted with one of frequent exacerbations of asthmatic bronchitis. Diagnose asthmatic bronchitis and released to go home. (EX 24).

12/26/01 – Emergency department report by Dr. Henzes – Examination showed lungs have decreased breath sounds with coarse rhonchi bilaterally. Chest x-ray showed changes consistent

with COPD, no true infiltrate is appreciated. Diagnoses: severe respiratory distress, COPD exacerbation. (EX 24).

12/26/01 – Examination by Dr. Kalis – Diagnosis: acute asthmatic bronchitis, rule out pneumonia superimposed on severe COPD, and other ailments. (EX 24).

12/26/01 – X-ray [portable] reading by Dr. Boyse – Patchy alveolar infiltrate visible in the right medial base. The left lung remains completely clear and there is no pleural effusion on either side. (EX 24).

12/26/01 – ABG by technician Davis – PCO₂ 64.8 / PO₂ 70.4. (EX 24).

12/26/01 – ABG by illegible technician – PCO₂ 54.8 / PO₂ 75.7. (EX 24).

12/26/01 – ABG by technician Mellon – PCO₂ 35.6 / PO₂ 84.6. (EX 24).

12/27/01 – Examination by Dr. Folke – Persistent patchy pulmonary infiltrate in the right lower lung similar to yesterday. The left lung is grossly clear. Diagnosis: condition consistent with pneumonia and COPD. (EX 24).

12/31/01 – X-ray reading by Dr. Mellon – Lungs are clear. (EX 24).

12/31/01 – X-ray reading by Dr. Mellon – Lung fields are clear. The cardiac silhouette, mediastinum, diaphragm, pleura, and bony thorax are normal. (EX 12).

1/1/02 – Discharge summary by Dr. Kalis – Chest x-ray showed a recurrent infiltrate of the right medial base. But at the time of discharge showed no wheezing, rales, and chest x-ray was clear. Diagnosis: Pneumonia and bronchitis in a patient with severe COPD. (EX 24).

1/9/02 – Examination by Dr. Kalis – Patient suffers from COPD with an exacerbation of pneumonia, but since release from hospital has improved. (EX 12).

1/10/02 – X-ray reading by Dr. Kalis – PA and lateral chest x-ray show the bony structures to be normal. The cardiac silhouette is unrevealing. The hilar are prominent bilaterally, but these shadows appear to be vascular in nature. No soft tissue or parenchymal infiltrates are noted. When compared to a previous film dated 11/7/01, there is no apparent change. No acute change. (EX 12).

3/2/02 – Emergency Department report by Dr. HENZES – Admitted for shortness of breath. Lungs show diffuse rhonchi bilaterally. X-ray shows borderline cardiomegaly, changes of his old COPD, no evidence of infiltrate or effusion. Diagnosis: COPD exacerbation. (EX 24).

3/2/02 – Examination by Dr. Kalis – No rales, rhonchi, or wheezes. Assessment: COPD with hopefully just mild exacerbation at this time, and other ailments. (EX 24).

3/2/02 – Discharge summary by Dr. Kalis – X-ray showed emphysema but no acute infiltrate. Patient was admitted with asthmatic bronchitis, which was with a mild exacerbation. Follow up x-ray was negative so he was discharged. Diagnosis: Asthmatic bronchitis and mild exacerbation. (EX 24).

3/2/02 – ABG by unknown technician – PCO₂ 38.5 / PO₂ 72.8. (EX 24).

3/4/02 – X-ray reading by Dr. Boyse – Emphysematous-appearing chest with no infiltrate. (EX 24).

3/4/02 – Letter from Dr. Kalis to discontinue as treating physician. (EX 19).

3/12/02 – Emergency department report by Dr. Popko – Admitted for shortness of breath. noted lungs have occasional expiratory wheeze, but no reduction in air exchange. Chest x-ray showed hyperinflation, but no acute process reported to the radiologist. Diagnosis COPD exacerbation. Discharged 3/13/2002. (EX 24).

3/12/02 – X-ray reading by Dr. Belk – Mild lung hyperaeration, no gross consolidation infiltrates are seen. (EX 24).

3/13/02 – Emergency department report by Dr. Popko – Admitted for shortness of breath. After his release, he saw primary physician and was sent back to emergency for reevaluation and admission. Chest x-ray shows no acute process. Diagnosis: COPD exacerbation. (EX 24).

3/13/02 – Examination by Dr. Mumma – Lungs hyperinflated and loud wheezes and evidence of severe air trapping bilaterally. Assessment: COPD, severe and increasing, and other ailments. (EX 24).

3/14/02 – Consultation by Dr. Branditz – He has coarse breath sounds bilaterally and moderate rhonchi. Chest CT scan showed emphysema changes with upper lung field bullae. There was no evidence of pulmonary embolism on that study. The patient has advanced emphysema and symptoms. He has chronic bronchitis and sputum. Some of this could be due to recurrent aspiration of airway secretions. (EX 24).

3/14/02 – Chest CT scan reading by Dr. Belk – Lung windows demonstrate multiple small lucencies involving the upper lobes due to COPD. No gross consolidative infiltrates or pleural effusions are identified. There is scarring noted at the right lung base. (EX 24).

3/14/02 – Examination by Dr. Din – Diagnosis: COPD. (EX 22).

3/17/02 – X-ray reading by Dr. Greenspan – Emphysematous chest without acute infiltrate. (EX 24).

3/17/02 – Consultation by Dr. Din– Lungs have occasional rhonchi, but otherwise good air entry. (EX 24).

3/18/02 – Left heart catheterization by Dr. Din. / Stint placement by Dr. Albrini. (EX 22).

3/20/02 – Stint placement by Dr. Albrini. (EX 22).

3/21/02 – Discharge summary by Dr. Albrini – Diagnosis: COPD. (EX 24).

4/30/02 – Follow-up examination by Dr. Albrini – Lungs: clear to auscultation. (EX 22).

5/30/02 – Examination by Dr. Din – Lungs: clear with occasional rhonchi. (EX 22).

6/13/02 – Emergency department report by Dr. HENZES – Admit for shortness of breath. Lungs had bilateral wheezing. X-ray showed lungs fully expanded, no evidence of infiltrate or effusion. Diagnosis: COPD exacerbation. (EX 24).

6/13/02 – Examination by Dr. Mumma – Assessment: exacerbation COPD, possible right lower lobe pneumonia given the physical findings and hypoxemia. (EX 24).

6/13/02 – X-ray reading by Dr. Muchnok – Subtle right posterior base infiltrate identified. No effusion is seen. Lung fields appear emphysematous and cardiac leads are identified overlying the chest. (EX 24).

6/13/02 - X-ray [portable] reading by Dr. Mellon – Lungs are clear, the pulmonary vascularity is normal, and the mediastinum, diaphragm and pleura have satisfactory appearance. (EX 24).

6/15/02 – Discharge summary by Dr. Metry – X-rays showed no evidence of pneumonia. Lungs showed minimal prolonged expiration with some fine end-expiratory wheezes. Assessment at admission was COPD exacerbation. (EX 24).

6/16/02 – Emergency department report by Dr. McCoy – Admitted for fever. Lungs have diminished breath sounds bilaterally with a few scattered expiratory wheezes. He does cough up some bright yellow sputum. X-ray shows lungs to be symmetrically well expanded, and infiltrate noted in the right lower lobe. Impression: pneumonia. (EX 24).

6/16/02 – Examination by Dr. Metry – Discharged on 6/15 because patient was doing well, had no abdominal pain, no shortness of breath, minimal wheezes. Pain reoccurred later that day and he came back in. X-ray show infiltrate, lungs show mild prolonged expirations and a few scattered wheezes. Diagnosis: Mild ileus with COPD, and other ailments. (EX 24).

6/16/02 – X-ray reading by Dr. Greenspan – Mild infiltrate at the right lung base which is similar in appearance to the study of 6/13/02. The left lung is clear. (EX 24).

6/17/02 – X-ray reading by D. Belk – Comparison is made with prior study performed on 6/15/02. There is mild lung hyperaeration with flattening of the hemidiaphragms. The previously described infiltrate at the right lung base apparently has resolved. No gross pleural effusions or pneumothorax is seen. (EX 24).

6/17/02 – CT Scan reading by Dr. Muchnok – Sections obtained from the lung base demonstrate a right lower lobe infiltrate that contains both an air space and interstitial component. (EX 24).

6/18/02 – Consultation by Dr. Pool – Lungs do demonstrate expiratory rhonchi with overall poor air movement. There were a few coarse inspiratory rales. Impression: dyspnea which may have multiple etiologies including COPD exacerbation but as well restenosis/coronary insufficiency, COPD, and other ailments. (EX 24).

6/20/02 – Left heart catheterization and stint placement by Dr. Albrini. (EX 22).

6/21/02 – Stint placement by Dr. Albrini. (EX 22).

6/23/02 – Discharge summary by Dr. Ahmad – Treated for COPD exacerbation and given antibiotics. (EX 24).

7/1/01 – Emergency Department report by Dr. McCoy – Admitted for chest pain. Lungs: clear to auscultation with a delayed expiratory phase noted bilaterally. The chest x-ray showed increased vascular markings on the right lower lobe. The left is otherwise clear. (EX 24).

7/1/02 – Examination by Dr. Mumma – Lungs are clear to auscultation, hyperinflated, and the lung fields are noted with mild crackles at the bases. Assessment: Chest pain considered and rule out unstable angina, Obstructive lung disease. (EX 24).

7/1/02 – Examination by Dr. Pool – Lungs: clear to auscultation. Impression: COPD and a variety of additional ailments. (EX 22).

7/1/02 – X-ray [portable] reading by Dr. Graber – Asymmetry of density in the lung fields. Left lung appears more lucent than the right. I understand the patient has a left hemiparesis and there may be diminished musculature on the left, which may account for the asymmetry and slightly more prominent musculature on the right. A discrete focal infiltrate is not definitely seen and the heart and mediastinal structures are intact. (EX 24).

7/1/02 – Discharge summary by Dr. Mumma – Labs are unremarkable. Diagnosis: Gastroesophageal reflux disease, atypical chest pain, obstructive lung disease, and other ailments. (EX 24).

8/13/02 – Examination by Dr. Din – Lungs: Clear with occasional rhonchi. (EX 22).

10/16/02 – Examination by Dr. Mumma – Lungs are clear to auscultation, chest x-ray reveals no evidence of acute congestive heart failure, pulmonary inflation, and otherwise, obstructive lung disease is present. There is no obvious infiltrate in the visual portions of the left chest. Assessment: History of emphysema and asthma and mild exacerbation of COPD. (EX 24).

10/16/02 – Consultation by Dr. Caffaratti – Lungs are generally clear with no audible wheezes, rales, or rhonchi. Assessment: Recurring dyspnea, which may represent an unstable angina

equivalent. Severe COPD is certainly exacerbation and could also account for dyspnea. Recommends cardiac catheterization so he can be definitive. (EX 24).

10/16/02 – X-ray reading by Dr. Mellon – There are mild chronic interstitial infiltrates at both lung bases, unchanged from 1/1/02 and 7/1/02. Lungs are clear of acute infiltrate. There is not pleural fluid. Conclusion – No active intrathoracic disease. (EX 24).

10/17/02 – Left heart catheterization by Dr. Caggaratti. (EX 22).

10/18/02 – Discharge summary by Dr. Mumma – Diagnosis: obstructive lung disease and other infirmities. (EX 24).

10/27/02 – Emergency department report by Dr. Stein – Lungs have diminished breath sounds in the bases, more diminished on the left than the right. Diagnosis: bronchitis and other ailments. (EX 24).

10/27/02 – X-ray reading by Dr. Boyse – Lungs are clear of any infiltrate, edema or pleural fluid. Chest is emphysematous with a low, flat diaphragm. (EX 24).

10/27/02 – ABG by technician Pitner – PCO₂ 40.7 / PO₂ 95.2. (EX 24).

11/6/02 – Emergency department report by Dr. Kerns – Lungs are diminished in all fields. Occasional wheeze is heard. 1:1 inspiratory and expiratory phase. Impression: No radiographic evidence of active disease. Acute COPD exacerbation, and acute bronchitis. (EX 24).

11/6/02 – X-ray reading – Lungs have an emphysematous or hyperinflated appearance. No acute infiltrate is localized when compared to a previous study of 10/27. Impression: emphysematous chest without acute findings radiographically. (EX 24).

11/12/02 – Examination by Dr. Din – Lungs: poor air entry though no wheezing or ronchi is noted. (EX 22).

11/19/2002 – PFT by Dr. Branditz – There is a severe obstructive lung disease pattern based on FEV₁ criteria. No significant change in expiratory flow rates after the administration of an inhaled bronchodilating agent. Inspiratory flow rates are normal. Lung volumes demonstrate an evaluation of total lung capacity and residual volume values. This could be consistent with some air-trapping from the above-noted obstructive lung disease state. Airways resistance values are significantly elevated, and specific airways conductance measurements are markedly decreased. These findings are also consistent with the above-noted findings; however, the values obtained seem out of proportion to the remainder of the study, and these values may be over-stated due to technical limitations of the study. The diffusing capacity for carbon monoxide is decreased. The decrease improves, but does not quite normalize when consideration is taken for simultaneously measured alveolar volumes. As noted above, the accuracy of the diffusing capacity result is unclear. (EX 24).

2/21/03 – Emergency department report by Dr. Blissenbach – Admitted for shortness of breath – Lungs are generally clear, no rales, a few scattered wheezes, breath sounds are equal. X-ray shows emphysematous changes, nothing acute. Re-exam shows clear lungs. Diagnosis: exacerbation of COPD – patient released. (EX 24).

2/21/03 – X-ray [portable] reading by Dr. Muchnok – The lung fields appear emphysematous. No infiltrate. (EX 24).

3/13/03 – Examination by Dr. Metry – Admission for hip. Mildly prolonged expiration in lungs. (CX 1).

3/31/03 – X-ray reading by Dr. Mellon – Granulomas about the hilar areas. The lungs are over penetrated but within its limitation appear clear of infiltrate. Conclusion: No active intrathoracic disease. (EX 23).

4/17/03 – Examination by Dr. Mumma – Assessment of respiratory effort reveals even respirations without use of accessory muscles and no intercostals retractions noted. Auscultation of lungs reveals clear lung fields and no rubs noted. Impression: Chronic cor pulmonale, Chronic airway obstruction. (EX 23).

5/6/03 – Consultation by Dr. Branditz – Lungs: prominent wheezing bilaterally, easily heard with a moderate pitch. There are no focal consolidated areas obvious. Chest x-ray was read free of infiltrate. Patient has a significant bronchospastic and somewhat bronchitic exacerbation of underlying COPD. (CX 1).

5/7/03 – X-ray reading by Dr. Greenspan – Lungs are hyperinflated. The present study is not as over penetrated in technique as the previous portable exam of 5/5. Interstitial markings are accentuated particularly on the right without signs of consolidation or effusion. Impression: Relative increase in interstitial density in lung fields. There may be some elements of atelectasis to the infiltrate at the right lung base since the right lung is not as hyperinflated as the left. (EX 23).

5/7/03 – Examination by Dr. Mumma – There is hyperinflation and loud wheezes and rales in all lung fields. He is slightly dyspneic at rest and extremely dyspneic with minimal exertion. Assessment: exacerbation of COPD and other ailments. (CX 1).

5/7/03 – X-ray reading by Dr. Greenspan – Relative increase in interstitial density in lung fields. There may be some element of atelectasis to the infiltrate at the right lung base since the right lung is not as hyperinflated as the left. (CX 1).

5/8/03 – X-ray reading by Dr. Boyse – Emphysematous chest with no infiltrate noted. (CX 1).

5/10/03 – X-ray reading by Dr. Belk – No acute abnormality detected. (CX 1).

5/19/03 – Examination by Dr. Mumma – Lungs reveal mild expiratory wheezes with excellent air movement bilaterally. Assessment: dyspnea manifesting as possible exacerbation of COPD, consider anginal equivalent, also consider panic attacks, other ailments. (CX 1).

5/19/03 – X-ray [portable] reading by Dr. Muchnok – Lung fields are emphysematous. A right base infiltrate is seen. The left lung is clear. (CX 1).

5/20/03 – Examination by Dr. Brantley – Lungs: markedly decreased breath sounds throughout, no rales, a prolonged expiratory phase of respiration. Impression: Asthmatic bronchitis, severe COPD/emphysema, no symptoms consistent with angina. (EX 22).

5/20/03 – Consultation by Dr. Branditz – Lungs have equal breath sounds, and only very mild expiratory wheezing. The patient has what sounds like a bronchitic or other infectious exacerbation of lung disease. This is diagnosed mostly by his purulent sputum production over the last couple of days. Sputum is decreasing at this time. He may have had bronchospasm early, but that is only very minimal at this time. (CX 1).

5/20/03 – X-ray reading by Dr. Graber – Mild peribronchial infiltrate, right middle lobe region, with a slight right basilar infiltrate. (CX 1).

5/22/03 – Discharge summary by Dr. Mumma – Diagnosis: End stage obstructive lung disease. (CX 1).

5/29/03 – Examination by Dr. Mumma – Chest inspection reveals chest hyperinflation, symmetric expansion and restriction due to osteoporotic changes. Assessment of respiratory effort reveals prolonged expiration bilateral, increase dyspnea with activity, pursed lip breathing and even respirations without use of accessory muscles. Chest percussion reveals resonance. Chest palpation reveals rhonchial fermitus. Auscultation of lungs reveals expiratory wheezes and no rubs noted. Impression: COPD with unspecific heart failure. (EX 23).

6/12/03 – Examination by Dr. Mumma – Assessment of respiratory effort reveals decreased diaphragmatic excursion, increased expiratory effort, hypertrophy of accessory respiratory muscles, and increase dyspnea with activity and even respirations without use of accessory muscles. Auscultation of lungs reveals clear lung fields and no rubs noted. Impression: Chronic airway obstruction NEC. (EX 23).

6/17/03 – X-ray reading by Dr. Greenspan – The lungs are clear to any infiltrate, edema, or pleural fluid. There is no sign of adenopathy. (EX 23).

7/17/03 – Examination by Dr. Mumma – Lungs are hyperinflated with moderate wheezes bilaterally. Assessment: COPD exacerbation, and other ailments. (EX 25).

7/17/03 – X-ray [portable] reading by Dr. Mellon – The lungs are symmetrically well expanded and clear. No significant pleural or parenchymal abnormality noted. (EX 25).

7/17/03 – ABG by illegible technician – PCO₂ 39.6 / PO₂ 95. (EX 25).

7/18/03 – Consultation by Dr. Basit – Chest x-ray showed normal. Chest is clear to auscultation. Assessment: stable with COPD, Mild shortness of breath due to mild bronchospasm in the ER. Lungs are clear. Saturations are excellent. (EX 25).

7/18/03 – Consultation: Dr. Feerick – Assessment: COPD. Chest x-ray is negative. (EX 25).

7/28/03 – Examination by Dr. Mumma – Assessment of respiratory effort reveals decreased diaphragmatic excursion, increased expiratory effort, hypertrophy of accessory respiratory muscles, and increase dyspnea with activity, and even respirations without use of accessory muscles. Auscultation of lungs reveals clear lung fields and no rubs noted. Impression: Chronic airway obstruction NEC. (EX 23).

9/4/03 – Examination by Dr. Mumma – Assessment of respiratory effort reveals decreased diaphragmatic excursion, increased expiratory effort, hypertrophy of accessory respiratory muscles, increased dyspnea with activity and even respirations without use of accessory muscles. Auscultation of lungs reveals clear lung fields and no rubs noted. Impression: Chronic airway obstruction NEC. (EX 23).

12/3/03 – Examination by Dr. Mumma – Could not auscultation carotid bruits due to inability to hold breath. He has accessory respiratory hypertrophy bilaterally. There is severe spastic paresis of his left upper extremity, which is mildly improved after being placed on Baclofen several weeks ago. He has bilateral wheezes, rales and adventitious lung sounds with no dullness present. Diagnosis: COPD with exacerbation. Increased exertional dyspnea. Mild cushing's syndrome secondary to chronic steroid for obstructive lung disease control. (CX 1).